

# Estimation of Serum Alkaline Phosphatase in Chronic Periodontitis in Smokers and Non-Smokers with Healthy Individuals: A Pilot Study

Nishant Agrawal<sup>1</sup>, Abhishek Singhvi<sup>2</sup>, Manoj Upadhyay<sup>3</sup>, Navneet Kaur<sup>4</sup>, Om Prakash Yadav<sup>5</sup>, Mohsin Khan<sup>6</sup>

1,3,4- Post Graduate, Department Of Oral Pathology, Vyas Dental College, Jodhpur, India.

2- Reader, Department Of Oral Pathology, Vyas Dental College, Jodhpur, India.

5,6- Post Graduate, Department of Public Health Dentistry, Rajasthan Dental College, Jaipur, India.

Correspondence to:

Dr. Nishant Agrawal,

Room No 14, Vyas PG Hostel, Vyas Campus, Near Kudi Haud, NH 65, Pali Road Jodhpur, 342005.

Contact Us : editor@ijdmr.com

Submit Manuscript : submissions@ijdmr.com

www.ijdmr.com

## ABSTRACT

**INTRODUCTION:** Tobacco smoking is recognized as an important environmental risk factor in periodontitis. It enhances the secretion of various enzymes from host cells which results in the initiation and progression of periodontal disease. Alkaline Phosphatase (ALP) is an enzyme found in the various cells of the periodontium is considered to causes destruction of periodontium. The aim of this study was to compare the ALP level in healthy individual with generalized chronic periodontitis in smokers and non smokers. **MATERIALS AND METHOD:** A total of 45 subjects were included in the study. The groups were divided in Group I (Generalized chronic periodontitis in smokers), Group II (Generalized chronic periodontitis in non-smokers) and Group III (Control group). The serum was analysed in the laboratory for ALP levels using a fully automated analyser. The results were statistically calculated with Student paired t-test. **RESULTS:** ALP level were higher in the subjects of chronic periodontitis in smokers habit when compared to non-smokers and healthy individuals. **CONCLUSION:** The present study conducted to evaluate and compare between the serums ALP levels in periodontitis with smokers, non smokers and in control group. We found that the ALP level of group I was significantly higher compared to group II and group III.

**KEYWORDS:** Chronic Periodontitis; Serum Alkaline Phosphatase; Smokers; Non-Smokers

## INTRODUCTION

Periodontitis is a chronic inflammatory disease of periodontium, which destroys the connective tissue and bone that supports teeth.<sup>1</sup> Periodontal disease is generally occurs due to an imbalance between pathogenic microbes and the local and systemic host responses. However, chronic periodontitis is known to vary by race, gender, and socioeconomic status, suggesting that factors related to the social environment may also have major role in terms of disease development.<sup>2</sup>

Cigarette smoking is a risk factor for many diseases,

and mounting evidence suggests that smoking adversely influences periodontal health. Recent data suggests that smoking has direct relation with periodontal health and independent to oral hygiene index. Many authors supported the role of smoking as a risk factor for periodontitis and stated that the potential risk reduces with cessation of smoking.<sup>3</sup> The response of an organism to the periodontal infection includes production of several enzymes and inflammation markers which can be analyzed both in serum as well as saliva. Recent studies have shown a correlation between high ALP levels and periodontitis, by proving the effect in

*How to cite this article:*

Agrawal N, Singhvi A, Upadhyay M, Kaur N, Yadav OP, Khan M. Estimation of Serum Alkaline Phosphatase in Chronic Periodontitis in Smokers and Non-Smokers with Healthy Individuals: A Pilot Study. Int J Dent Med Res 2014;1(4):30-34.

periodontal diseases.<sup>4</sup> ALP is a membrane bound glycoprotein found on cell membrane. It is released from poly-morphonuclear neutrophils during inflammation, osteoblast during bone formation and periodontal ligament fibroblast during periodontal regeneration.<sup>5</sup> Aim of this study was to compare the ALP level in healthy individual with patients having chronic periodontitis with the habit of smoking and patients having chronic periodontitis without the habit of smoking. The activity level of this enzyme could be reflected at the serum level and this depicts a correlation between chronic periodontitis and ALP level.

## MATERIALS & METHODS

The study was conducted in the Department of Oral Pathology of Vyas Dental College and Hospital, Jodhpur among the patients visiting the Department of Periodontics of the same college. A total of 45 subjects aged between 30 and 65 years were included in this study. Of these subjects 15 had smoking associated chronic periodontitis (Group I) and 15 were chronic periodontitis devoid of smoking (Group II). For comparison 15 healthy individuals were also studied for serum ALP level (Group III).

For clinical examination minimum 18 teeth in each subject were examined for the depth of periodontal pocket with the help of William's periodontal probe. A minimum of one pocket with probing depth of 6 mm or above was considered as chronic periodontitis. The patients suffering from systemic conditions like diabetes, cardiac disease and hypertension were excluded because in these diseases the pathophysiology for periodontitis was altered.

2 ml of venous blood was drawn by venipuncture. Blood was collected in the test tube which was allowed to stand for 30 minutes at room temperature and centrifuged at 3000 rpm for 5 minutes and separate serum from it. Serum was analyzed using COBAS INTEGRA 400 plus fully auto analyzer to estimate ALP. Data was calculated for mean and standard deviation. The statistical significance of

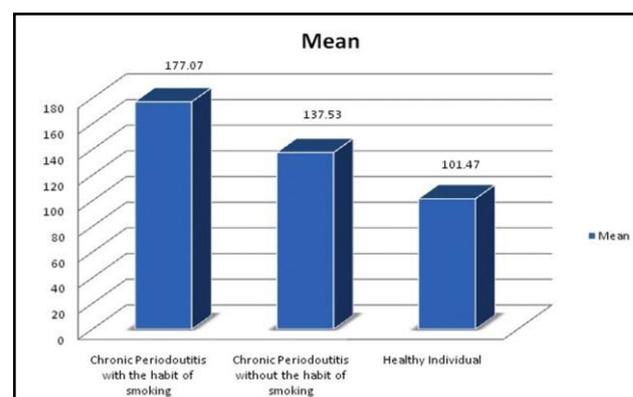
difference between groups was tested with student paired T-test.

## RESULTS

The study revealed that ALP level in group I ranges between 227 micron/ml to 151 micron/ml. group II exhibiting 171.7 micron/ml to 101.7 micron/ml serum ALP level. Control group had lowest range of serum ALP concentration which range between 110 micron/ml to 101.1 micron/ml. The mean of serum ALP level was highest in the group I, which was 177 micron/ml compares to other two groups. The group II showing higher mean value ( 137.5 micron/ml) compares to control group ( 101.5 micron/ml) (Table 1, Graph 1).

Groups	N	Sum	Mean	Std. Deviation
Chronic Periodontitis with the habit of smoking	15	2656	177.07	27.763
Chronic Periodontitis without the habit of smoking	15	2063	137.53	15.151
Healthy Individual	15	1522	101.47	14.510

Table 1 showing mean and Std. Deviation of different study groups



Graph 1 showing mean value of different study groups

Statistical analysis revealed a significant difference between Group I and Group II ALP level. These both groups also have significantly higher values of serum ALP level with compare to control group. (Table 2)

Groups	P- value < 0.05
Chronic Periodontitis with the habit of Smoking and Chronic Periodontitis without the habit of Smoking	0.001
Chronic Periodontitis with the habit of Smoking and Healthy Individual	0.001
Chronic Periodontitis without the habit of Smoking and Healthy Individual	0.001

Table 2 Showing P Value Between Different Groups

## DISCUSSION

Periodontitis is a chronic inflammatory condition characterized by loss of connective tissue and alveolar bone with the formation of periodontal pockets due to the complex interaction between pathogenic bacteria and the host's immune response.<sup>6</sup> Subjective symptoms of periodontitis are typically mild during progression of the disease. Usually subjects ignore the condition in early phase until more severe symptoms (such as tooth mobility) appear.<sup>7</sup> The response of an organism to the periodontal infection includes production of several enzymes and inflammation markers which can be analyzed both in serum and saliva.<sup>8</sup> In periodontitis, one of the mechanisms of collagen loss is fibroblast phagocytize collagen fibers which contributes to the total ALP level.<sup>9</sup> ALP is found primarily in the liver (isoenzyme ALP 1), in the bone (isoenzyme ALP 2) and small amount produced by cell lining the intestine (isoenzyme ALP 3), the placenta and the kidney. The amount of ALP in the blood is calculated and represented as the total amount of ALP released from the tissues.<sup>10-12</sup>

Among the host enzymes, the first one identified one was ALP. It is most effective in an alkaline environment. The optimal pH for the activity of ALP is 8.0-8.5 depending on the source.<sup>13</sup> ALP is stored in specific granules as well as secreted by vesicles of neutrophils and is mainly released during their migration to the site of infection.<sup>14</sup> Ishikawa and Cimasoni identified the potential of ALP as an important biochemical marker of gingival crevicular fluid (GCF).<sup>10</sup> Increased in the inflammation and

bone turn over rate can cause increased activity of ALP. This is probably a consequence of pathological processes in periodontal tissues as ALP is produced by PMNs, osteoblasts, macrophages, fibroblasts and plaque bacteria within periodontal tissue or periodontal pocket.<sup>15</sup> Further more, among the various periodontopathogenic bacteria like *Prevotella intermedia* and *Porphyromonas gingivalis* are known to have high ALP activity and in this study the smoker group was shown to have higher plaque index which results in high ALP level.<sup>16</sup>

Cigarette smoking is associated with periodontal disease and considered as major risk factor that lead to increased severity of periodontal disease, rates of disease progression and this destruction of periodontal tissue in smokers.<sup>17</sup> It was observed that the development of gingival redness was lower in smokers suggesting a suppression of the normal inflammatory response to plaque and calculus. The reduced gingival bleeding due to the potential vasoconstrictive effects of nicotine may lead to misdiagnosed of periodontitis.<sup>18,19</sup> The vasoconstriction of peripheral blood vessels which occurs due to smoking can also affect the periodontal tissue and can lead to less overt signs of gingivitis such as redness, bleeding and exudation.<sup>19</sup> Smoking results in increased periodontal destruction by altering the host response through impairment of the normal host response in neutralizing infection and alteration results in destruction of the surrounding periodontal tissues.<sup>20</sup>

A number of studies have shown raised serum ALP levels in various physiological and pathological conditions like bone growth physiologically and bone diseases. ALP showed a significant rise in both diabetic and non-diabetic patients with periodontitis as compared to control.<sup>21</sup> Raised value of serum ALP in periodontitis has been reported by Grossi, 1998; Iwamoto et al, 2001; Siddiqui et al, 2005 same as our study.<sup>22</sup> Most of the studies are related to levels of ALP in GCF of patients with periodontal disease. Very few studies have evaluated serum ALP levels in patients with chronic periodontitis.<sup>3,22</sup>

Gibert in 2003 predicted ALP as an indicator for future loss of periodontium. It may serve as marker in periodontal treatment planning and monitoring. Its level may also be useful as a potential bone turnover marker to establish the diagnosis and prognosis of periodontal disease.<sup>23</sup> Binder et al demonstrated that ALP concentration in GCF showed a positive relationship with attachment loss.<sup>12</sup> Gao et al. in 1999 found that ALP activity was highest in osteoblasts, moderate in periodontal ligament fibroblast and lowest in gingival fibroblast.<sup>24</sup> Other biochemical markers like total cholesterol, high density lipoprotein, low density lipoprotein, LDH etc were measured, but as stated earlier ALP is the first marker which identified in blood among all other markers in the periodontitis.<sup>8</sup> ALP is essential enzyme, because it is part of normal turnover of periodontal ligament, root cementum and bone hemostatis.<sup>24</sup>

The present study showed that serum ALP levels were positively correlated with all clinical parameters. This increased activity in the serum is probably the consequence of destructive process in the alveolar bone in periodontitis. This was in agreement with studies done by Nakumura M and Slots J, Gibert P et al, Totan A, Greabu M, Totan C et al, Todorovic T, Dozic I, Vicente-Barrero M et al who compared the enzyme activities in subjects with periodontitis and in healthy controls. They found that periodontal destruction such as periodontal pocket, gingival bleeding and suppuration were related to higher ALP levels and concluded that the increase in serum ALP activity in periodontitis could be associated with alveolar bone loss, a key feature of periodontitis.<sup>19,25-27</sup>

The results of the present study indicate the positive correlation between the periodontitis patients having habit of smoking and serum ALP activities. The tendency of linear increase in the level of ALP activity in serum reflects the advancing periodontal tissue injury and damage. Further studies in this direction with larger study sample and especially longitudinal clinical studies involving the post periodontal therapy assessment of serum ALP in comparison to the pre treatment activity levels could

prove of immense help in the management of periodontitis in the near future.

## CONCLUSION

The present study was conducted to evaluate and compare between the serum ALP levels in chronic generalized periodontitis with smokers, non smokers and healthy controls. Though there are many studies regarding detection of levels of ALP in GCF and saliva but in serum there are very few. ALP levels can be detected in serum of patients with periodontitis. There was a highly significant correlation of increased levels of serum ALP in chronic periodontitis as compared to healthy patients. There was a highly significance correlation of high levels of serum ALP in chronic periodontitis with smokers and non-smokers.

## REFERENCES

1. Newman et al. Carranza's Clinical Periodontology. 10<sup>th</sup> edi. Elsevier publication. 2006, pp 434.
2. Kibayashi M et al. Longitudinal study of the association between smoking as a periodontitis risk and salivary biomarkers related to periodontitis. J Periodontal. 2007; 78 (5): 859 – 67.
3. Sanikop S et al. A comparative analysis of serum alkaline phosphatase in smokers and non-smokers with chronic periodontitis. SRM University J of Dental Science. 2011; vol. 2 (4): 290-95.
4. Haber J et al. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontal 1993;64:16-23.
5. Nanci A et al. Ten Cate's Oral histology Development, structure and function. 7<sup>th</sup> edition. Elsevier publication. 2013. Pp 236.
6. Armitage G. clinical evaluation of periodontal diseases. Periodontal 2000 1995;20:1183-97.
7. Berglundh T et al. aspect of adaptive host response in periodontitis. J Clin Periodontal 2005;32:87-107.
8. Gopinath et.al. International journal of applied biology and pharmaceutical technology. 2010; vol. 1 (3) : 1202 – 08.
9. Robbins and Cotran. Pathologic basis of disease, 7<sup>th</sup> edition Elsevier publication.
10. Ishikawa et al. alkaline phosphatase in human gingival fluid and its relation to periodontitis. Arch Oral Biol 1970;15:1401-4.

11. Shibata Y et al. effective method of discriminating between oral bacterial and human alkaline phosphatase activity Oral Microbiol Immunol 1994;9:35-9.
12. Binder TA et al. gingival fluid levels of acid and alkaline phosphatase. J Periodontal Res 1987;22:14-9.
13. Perinetti G et al. gingival crevicular fluid alkaline phosphatase activity reflects periodontal healing/recurrent inflammation phases in chronic periodontitis patients. J Periodontal 2008;79:2000-7.
14. Daltabano et al. gingival crevicular fluid alkaline phosphatase levels in postmenopausal women: effect of phase I periodontal treatment J Periodontal 2006;7:453-65.
15. Chapple E et al. analysis of saliva for periodontal diagnosis- a review J CLin Periodontal 2000;27:453-65.
16. Ellis E et al. The role of alkaline phosphatase in mineralization. Curr Opin Orthop 2007;18:444-8.
17. Karem B et al. Periodontal health status and salivary enzymes level in smokers and non-smokers (comparative, cross sectional study). J Bagh Coll Dentistry 2012;25(3):91-6.
18. Perinetti G et al. alkaline phosphatase activity in gingival crevicular fluid during human orthodontic tooth movement Am J Orthod Dentofacial Orthop 2002;122:548-56.
19. Todorovic T et al. salivary enzymes and periodontal diseases. Med Oral Patol Oral Cir Bucal 2006;11:E115-9.
20. Wolf BH et al. multivariate analysis of oral hygiene data from a representative sample. J Clin Periodontal 2001;28:891-4.
21. Shaheen A et al. Serum alkaline phosphatase level in patients with type 2 diabetes mellitus and its relation with periodontitis. KUST Med J 2009;1(2):51-4.
22. Sakalauskiene J et al. Alkaline phosphatase activity changes of blood neutrophil leukocytes among patients suffering from diabetes mellitus type I and periodontal diseases. J Stom, Balt Denta Maxillofacia 2004;6(2):55-8.
23. Malathi K et al. Salivary enzymes- a diagnostic marker for periodontal disease. IJSRR 2012;2(4):68-80.
24. Gao J et al. should cementoblasts express alkaline phosphatase activity? Preliminary study of rat cementoblasts in vitro. J Periodontal 1999;70(9):951-9.
25. Nakamura M et al. salivary enzymes: origin and relationships to periodontal diseases. J Periodontal Res 1983;18:559-69.
26. Gibert P et al. alkaline phosphatase isozyme activity in serum from patients with chronic periodontitis J Periodontal Res 2003;38:362-5.
27. Tutan A et al. salivary AST, ALT and ALP; possible markers in periodontal diseases? Clin Chemistry Lab Med 2006;44:612-5.

Source of Support: Nil

Conflict of Interest: Nil