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EVALUATION OF SERUM ALBUMIN AND OTHER BIOCHEMICAL PARAMETERS IN PATIENTS WITH CHRONIC PERIODONTITIS

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ABSTRACT

Chronic periodontitis is an inflammatory disease of the oral cavity resulting in inflammation within the supporting tissue of the teeth with progressive clinical attachment and bone loss. It is most prevalent form of periodontitis. It is observed that in presence of systemic disease like DM etc which influences the host defense mechanism the progression of the disease is more aggressive. This study was undertaken to evaluate serum albumin levels which is a practical marker of general health status. Since albumin is synthesised in the liver, presence of any liver abnormality is also detected by measuring the serum level of enzymes like alanine transaminase (ALT), aspartate transaminase (AST) and alkaline phosphatase (ALP). It has been suggested that liver abnormality could make individual more susceptible to periodontal disease. The present study included 80 subjects, 40 were chronic periodontitis patients and 40 were healthy subjects. The subjects in the study and control group were aged between 30 to 60 years. The biochemical parameters which were evaluated were serum albumin, serum al alanine transaminase, aspartate transaminase and alkaline phosphatase. It was found that serum albumin levels were significantly lower in chronic periodontitis patients than healthy subjects. Inverse association was seen between serum albumin and clinical attachment loss in chronic periodontitis patients. Serum ALT and ALP showed significant increase in patients with chronic periodontitis as compared to healthy subjects. This study was undertaken to evaluate the role of serum albumin and liver enzymes in the cause effect relationship of chronic periodontitis.

KEYWORDS: Chronic periodontitis, Serum albumin, Alanine transaminase, Alkaline Phosphatase.

INTRODUCTION

Chronic periodontitis is the disease of the oral cavity consisting of chronic inflammation of periodontal tissues, that is caused by accumulation of profuse amount of dental plaque characterised by progressive clinical attachment and bone loss.[1] Clinically patient presents with redness and swelling of gums which bleeds on probing, loss of tooth. Periodontal disease is the component of global burden of chronic disease. The prevalence rate of periodontitis is high in India, affecting more than 50% of the Indian community.[2] Severe periodontal disease is due to poor oral hygiene or poor general health status although it has multiple risk factors including microbial, behaviourial and systemic factors. Presence of systemic diseases like diabetes mellitus increases the severity and the extent of periodontitis since it influences the effectiveness of the host immune response. Periodontal infection in itself has been implicated as a risk factor for systemic diseases like cardiovascular disease, diabetes mellitus.[3] It is suggested that impaired dentition due to periodontitis may affect individual by causing dietary restrictions compromising the general health of the individual leading to malnutrition.[4] In periodontitis bacteria triggers inflammatory host responses and factors such as malnutrition diminishes host immune response like phagocytic function,complement system etc. thus leading to progression of the disease.[5] Serum albumin is considered as a marker of general health status and therefore malnutrition may be monitored by measuring the serum level of albumin. Albumin is the major component of plasma protein, helps in maintaining the plasma colloid osmotic pressure. Normal level is between 3.5 to 5.0 g/dl. It is synthesised by the liver. Inflammation and malnutrition both reduces the synthesis of albumin in the body. This study is done to evaluate the association between serum albumin and chronic periodontitis and whether serum albumin levels are affected by inflammatory or nutritional component of chronic periodontitis. Along with serum albumin levels, biochemical parameters like SGOT/AST, SGPT/ALT, ALP, were also estimated to determine whether periodontal disease is associated with liver abnormalities. As a systemic factor, hepatic conditions have suggested a positive association with periodontal disease.[6] A study in Japan reported that individuals with periodontal disease had elevated levels of liver enzymes, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST).[7] Amino transferase is an enzyme that is a catalyst in the transfer of $-NH_2$ group from L-amino acid to keto acid creating new amino acid and keto acid. Alanine

Amino transferase is primarily found in hepatocyte intracellular fluid.[8] Serum levels are used to detect hepatic distress and diseases such as chronic viral infections, autoimmune hepatitis, and steatohepatitis. Many mechanisms and pathways may be involved in elevated serum Alanine Aminotransferase levels; however, chronic inflammation is one potential mechanism for its increase. Alkaline phosphatase is hydrolytic enzyme acting optimally at pH 10. Various enzymes are released from host cells during the initiation and progression of periodontal disease.[9,10] Alkaline Phosphatase (ALP) is an enzyme found in cells of the periodontium, including osteoblasts, fibroblasts, and neutrophils. Studies show that concentrations of this enzyme in gingivo-crevical fluid (GCF) from diseased sites are significantly higher than from healthy sites. Studies have associated whole mouth ALP levels with the progression of periodontitis.[11,12] The injured tissue secrete alkaline phosphatase from the neutrophils that causes destruction of connective tissues and level of ALP activities directly correlate with the intensity of inflammatory process of periodontal tissue.[13] The purpose of this study is to find if there is any association between liver abnormalities and chronic periodontitis.

MATERIALS AND METHODS

Selection of subjects

A total of 80 subjects of both genders with age range of 30 to 60 years were included in this study. Patients were selected from the OPD of Department of Periodontics,Goa Dental College, bambolim Goa. The informed consent of the subjects was taken after obtaining ethical clearance from institution's ethical committee. Detailed medical and dental history was taken prior to inclusion into the study and control groups. The selected subjects were divided into two groups: groupI included 40 subjects with chronic periodontitis with clinical attachment loss of >5mm and group II included 40 healthy subjects. Patients with history of viral hepatitis, cirrhosis, liver cancer, cholecystitis were excluded.

Periodontal examination: was performed on all 80 subjects by the dentist. Clinical indices of periodontal disease which were calculated included gingival index score, mean probing depth, mean attachment loss for all the present teeth. This study focussed the analysis on clinical attachment loss which is a measure of periodontal tissue lost because of the disease process. It was measured using periodontal probe. Attachment loss of >5mm were used as a case definition of periodontitis.

For biochemical analysis 5 ml of blood was drawn from antecubital vein under aseptic conditions. Thereafter serum and was separated by centrifugation at 3000 rpm in clinical centrifuge for 10 minutes. Serum was used for measurement of albumin, alanine transaminase, aspartate transaminase, alkaline phosphatase and total protein. All the estimations were done within 24 to 48 hours after specimen collection in the clinical laboratory of department of biochemistry, Goa Medical College, Bambolim. These tests were done on Architect ci 8000 clinical chemistry autoanalyser by Abott Diagnostics.

SERUM ALBUMIN WAS DETECTED BY BROMOCRESOL GREEN METHOD.[14]

The Albumin BCG procedure is based on the binding of bromcresol green specifically with albumin to produce a colored complex. The absorbance of the complex at 628 nm is directly proportional to the albumin concentration in the sample.

SERUM ALANINE TRANSAMINASE BY IFCC METHOD[15]

ALT present in the sample catalyzes the transfer of the amino group from L-Alanine to 2-Oxoglutarate, in the presence of Pyridoxal5'-Phosphate, forming Pyruvate and L-Glutamate. Pyruvate in the presence of NADH and Lactate Dehydrogenase (LD) is reduced to L-Lactate. In this reaction NADH is oxidized to NAD. The reaction is monitored by measuring the rate of decrease in absorbance at 340 nm due to the oxidation of NADH to NAD.

SERUM ALKALINE PHOSPHATASE BY PARA NITRO PHENYL PHOSPHATE METHOD[16]

Alkaline phosphatase in the sample catalyzes the hydrolysis of colorless p-nitrophenyl phosphate (p-NPP) to give p-nitrophenol and inorganic phosphate. At the pH of the assay (alkaline), the p-nitrophenol is in the yellow phenoxide form. The rate of absorbance increase at 404 nm is directly proportional to the alkaline phosphatase activity in the sample. Optimized concentrations of zinc and magnesium ions are present to activate the alkaline phosphatase in the sample

SERUM ASPARTATE TRANSAMINASE BY NADH METHOD[17]

AST present in the sample catalyzes the transfer of the amino group from L-aspartate to 2-oxoglutarate, in the presence of pyridoxal-5'phosphate, forming oxaloacetate and L-glutamate. Oxaloacetate in the presence of NADH and malate dehydrogenase (MDH) is reduced to L-malate. In this reaction, the NADH is oxidized to NAD. The reaction is

monitored by measuring the rate of decrease in absorbance at 340 nm due to the oxidation of NADH to NAD.

Table 1: Distribution of Total Subjects

Group	Number of cases
Chronic periodontitis	40
Controls	40
Total	80

Table 2: Table showing mean levels of Several parameters between chronic periodontitis patients and control group

Group	No. of cases	Serum Albumin(g%)	Serum ALT(U/L)	Serum AST (U/L)	Serum ALP(U/L)
Chronic periodontitis	40	4.0±0.29	74±3.14	18±1.56	155±1.22
Controls	40	4.4±0.3	33±2.5	21±2.2	64±3.3

Table 3: Table showing mean levels of clinical attachment loss(CAL),gingival index(GI) score and probing depth(PD) among chronic periodontitis patients and control group

Group	No. of cases	CAL(mm)	PD(mm)	GI score
Chronic periodontitis	40	1.67±0.81	2.00± 0.29	1.15±0.53
Controls	40	0.00±0.00	1.06± 0.08	0.02±0.019

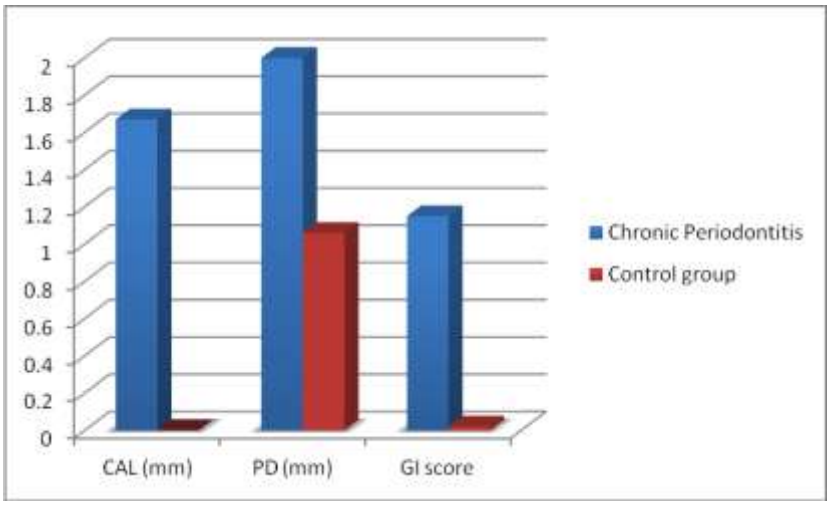

Figure 1: Mean levels of clinical attachment loss(CAL),gingival index(GI) score and probing depth(PD) among chronic periodontitis patients and control group

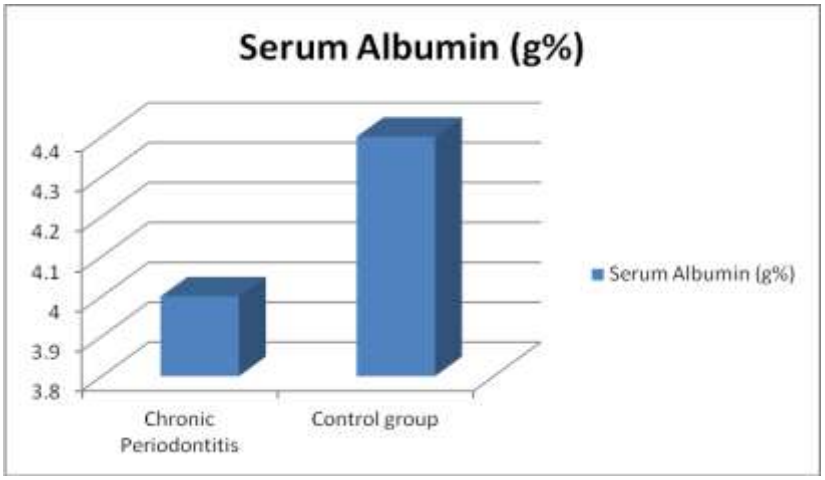

Figure 2: Mean levels of serum albumin among chronic periodontitis patients and control group

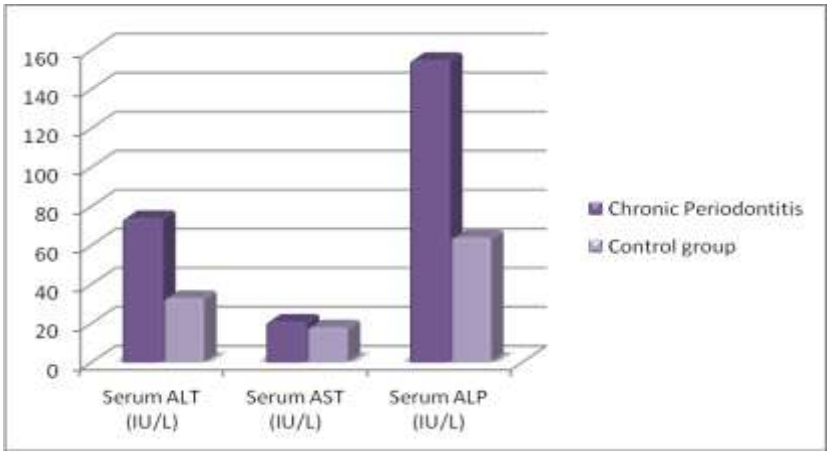

Figure 3: Mean levels of parameters like serum ALT, AST and ALP between chronic periodontitis patients and control group

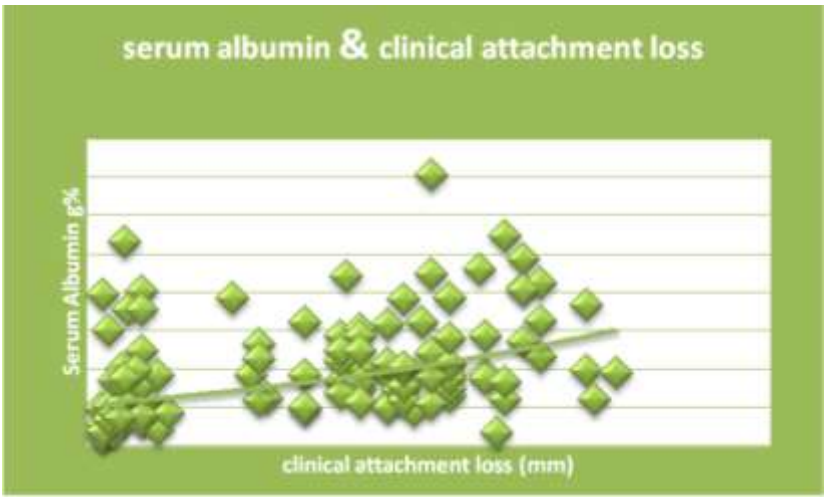

Figure 4: Correlation between albumin and clinical attachment loss in periodontitis group

RESULTS

This study consisted of 80 subjects, group I included 40 healthy subjects and group II included 40 chronic periodontitis patients with clinical attachment loss of >5mm. The mean serum albumin level for group I was 4.0 with standard deviation of 0.30 and group II was 4.4 with standard deviation of 0.3.

On comparison of both the values using the Students′ unpaired ′t′ test, the differences between the serum albumin levels in Group I and Group II were found to be statistically significant. The clinical periodontal indices values were grossly different between both the groups. The mean clinical attachment loss for group I was 0.00 and for group II was 1.67 with standard deviation of 0.81. The mean probing depth for group I was 1.06 with standard deviation of 0.080 and group II was 2.00 with standard deviation of 0.29. The mean gingival index score for group I was 0.02 with standard deviation of 0.019 and group II was 1.15 with standard deviation of 0.53. All the three clinical indices showed significant difference between test and the control groups i.e. sig. (2 tailed) p value <0.05. The mean serum AST level in group I was 18 IU/L and group II was 21 IU/L which showed no statistical significant difference between the two groups. Similarly serum ALP level was determined in the two groups with a mean value of 155 U/Lin chronic periodontitis patients and 64U/L in healthy subjects. The mean of serum ALT in group I was 33 IU/Land group II was 74 IU/L which was found to be statistically significant. Correlation between clinical attachment loss and serum albumin was calculated in the test and control group. The inverse correlation between the two parameters was seen in chronic periodontitis group.

DISCUSSION

In the present study it was seen that serum albumin is significantly low in chronic periodontitis patients as compared to healthy subjects. These findings are similar to studies like ogawa et al[18] in which inverse association was found between serum albumin and chronic periodontitis.it is difficult to infer that the decreased level of serum albumin is due to inflammatory component of chronic disease or due to poor nutritional status secondary to poor dentition. It has been suggested that impaired dentition such as tooth loss in periodontitis may affect the individual causing diet restrictions i.e. difficulty in chewing compromising the nutritional states and well being.

In our study possibility of nutritional status affecting the serum albumin was ruled out since the subjects included in the trial had a mean dentate percentage of 28 teeth per subject.

Therefore it is inferred that low serum albumin is because of chronic inflammation of the periodontal tissue. Similar findings were reported by Yoshihara et al[19] and Rajashree et al.[20]

Study by Saravan et al[21] showed significant association between serum albumin and chronic periodontitis in elderly patients.subjects showed decreased albumin level with increase in loss of clinical attachment of >6mm. According to Hermann et al[22] inflammatory states, liver diseases,renal diseases have been indicated to reduce serum albumin levels. Study by pimpale parmar et al[23] suggested that periodontitis results in formation increased levels of C reactive protein, this stimulates antibody formation like Ig G. Because of increased formation of C reactive protein and IgG by the liver in diseased conditions, albumin levels are decreased in serum. Iwaski et al[24], found significant association between periodontitis disease and serum albumin levels. Our study also showed inverse correlation i.e. negative association between serum albumin and clinical attachment loss in the chronic periodontitis group. This shows that as the clinical attachment loss increases there is significant reduction in the serum albumin levels in chronic periodontitis patients. Thus serum albumin can serve as an indicator of severity of chronic periodontitis condition. Similar findings were reported by Ramesh Amitha et al.[25]

Since periodontitis is an inflammatory disease resulting in destruction of soft and hard oral tissues supporting the teeth, the biochemical markers like enzymes play a crucial role in detecting the inflammatory changes taking place in the tissue. Intracellular enzymes like AST, ALT, ALP, CK (creatine kinase) are released from periodontal tissue into gingival cervicular fluid and saliva. They can serve as a important parameters for diagnosis, treatment and prognosis of periodontitis.[26] The present study was done to evaluate if there is any changes in the liver enzymes such as AST, ALT and ALP associated with chronic periodontitis disease. It was found that there is significant increase in the levels of serum ALT and ALP in chronic periodontitis patients. Serum levels of AST did not show significant difference in both the groups and was found to be within normal limits. Study by R Constance Weiner et al[27], showed significant association between serum ALT and chronic periodontitis. It has been suggested that changes in enzymatic activity indicate metabolic change in inflammation and that Alanine Aminotransferase release increases with cell injury and death. Thus increased level of serum ALT indicates the severity of the disease. Researchers in a study of 49 older adult Japanese patients with chronic periodontitis found

Alanine Aminotransferase to be associated with periodontal inflammation.[28] Salivary Alanine Aminotransferase was increased in individuals with periodontitis disease, as compared with controls in a study of 40 patients in India.[29] Increased ALT could be a risk indicator thus monitoring of hepatic abnormalities can be done to prevent periodontitis. ALP showed a significant rise in both diabetic and non-diabetic patients with periodontitis as compared to controls.

CONCLUSION

The present study showed significant reduction in serum albumin levels in chronic periodontitis patients.there was significant inverse association between serum albumin and clinical attachment loss in patients with chronic periodontitis.also found that there was significant increase in serum ALT and ALP levels in chronic periodontitis patients as compared to healthy subjects. Such a study will help the clinician for dental referral of the patient with high ALT and ALP levels.

REFERENCES

1. Newman, Takei, Carranza. Chronic periodontitis. Clinical Periodontology.9th edition. New Delhi. Harcourt private limited, 2003; 398.
2. Shah N. National Commission on Macroeconomics and Health, Ministry of Health and Family Welfare. New Delhi: Government of India. Oral and dental diseases: Causes, prevention and treatment strategies In NCMH Background Papers—Burden of Disease in India (New Delhi, India), Sep, 2005; 275–98.
3. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiological perspective. Ann Periodontol, 2001; 6: 99–112.
4. Chauncey HH, Muench ME, Kapur KK, Wayler AH. The effect of the loss of teeth on diet and nutrition. Int Dental J., 1984; 34: 98–104.
5. Enwonwu CO, Phillips RS, Falkler WA. Nutrition and oral infectious diseases: state of the science. Compend Contin Educ Dent., 2002; 23: 431–4,436,438.
6. Saito T, Shimazaki Y (2007). Metabolic disorders related to obesity and periodontal disease. Periodontol, 2000; 43: 254-266.
7. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between periodontitis and hepatic condition in Japanese women. J Int Acad Periodontol, 2006; 8: 89-95.

8. Z. Liu, S. Que, J. Xu, and T. Peng, "Alanine aminotransferase-old biomarker and new concept: a review," International Journal of Medical Sciences, 2014; 11(9): 925–935.
9. Renggli HH: Phagocytosis and killing by cervicular neutrophils. In Lehner T (ed): Periodontal disease and diabetes mellitus. New York, Grune and Stratton, 1977.
10. Palmer J.P, Culloch M C. Prediction and prevention of IDDM. Diabetes, 1982; 40: 943-7.
11. De Bernard B. Glycoprotiens in the local mechanisms of calcification. J Clin Orthop, 1982; 162: 233-44.
12. Safkon-Seppala B, Ainamo J. Periodontal condition in insulin dependent diabetes mellitus. J Clinical periodontal, 1992; 19: 24-9.
13. Aiinamo J, Lahtinen A, Uitto V. Rapid periodontal destruction in adult human with poorly controlled diabetes. J Clin Periodontal, 1990; 17: 22-8.
14. Architect, Aeroset. abott clinical chemistry. Albumin BCG package insert. abott laboratories, 2007.
15. Bergmeyer HU, Horder M, Rej R. Approved recommendation (1985) on IFCC methods for the measurement of catalytic concentration of enzymes. J Clin Chem Clin Biochem, 1986; 24(7): 481–95.
16. Bessey OA, Lowry OH, Brock MJ. A method for the rapid determination of alkaline phosphatase with five cubic millimeters of serum. J Biol Chem, 1946; 164: 321–9.
17. Bergmeyer HU, Horder M, Rej R. Approved recommendation (1985) on IFCC methods for the measurement of catalytic concentration of enzymes. J Clin Chem Clin Biochem, 1986; 24(7): 497–510.
18. Ogawa H, Yoshihara A, Amarasena N, Hirotomi T, Miyazaki H. Association between serum albumin and periodontal disease in community-dwelling elderly. J Clin Periodontol, 2006; 33: 312–6.
19. Yoshihara A, Hanada N, Miyazaki H. Association between serum albumin and root caries in community-dwelling older adults. J Dent Res., 2003; 82: 218–22.
20. Rajashri A. Kolte et al. Quantitative estimation and correlation of serum albumin levels in clinically healthy subjects and chronic periodontitis patients. J Indian Soc Periodontol, Oct-Dec, 2010; 14(4): 227–230.
21. S aravanan AV et al. Estimation of Serum Albumin Levels associated with Chronic periodontitis in Elderly Subjects - A Cross Sectional Study. SRM University Journal of Dental Sciences, April–June, 2012; 3(2).

22. Francois R. Herrmann, Charles Safran, Sue E. Levkoff and Kenneth L. Minaker. Serum Albumin Level on Admission as a Predictor of Death, Length of Stay, and Readmission. Arch Intern Med., January, 1992; 152(1): 125130.
23. Sandeep K. Pimpale, Parmar et al. Evaluation of the relationship between periodontal disease and general health status in chronic periodontitis patients using serum albumin concentration. International Journal of Dental and Health Sciences, 01(04): 468-474.
24. Iwasaki M, Yoshihara A, Hirotomi T, Ogawa H, Hanada N, Miyazaki H. Longitudinal study on the relationship between serum albumin and periodontal disease. J Clin Periodontol. 2008; 35: 291–6.
25. Ramesh Amitha, John Anju Mary et al. Association of serum albumin concentration and periodontitis.international research journal of Pharmacy, 2012; 3(11): 183-186.
26. American Academy of Peridontology. Position Paper: Diagnosis of periodontal diseases. J Periodontol, 2003; 74: 1237–47.
27. R. Constance Wiener. Association of Alanine Aminotransferase and Periodontitis: A Cross-Sectional Analysis—NHANES, 2009–2012.
28. H. Yoshie, H. Tai, T. Kobayashi et al., "Salivary enzyme levels after scaling and interleukin-1 genotypes in Japanese patients with chronic periodontitis," Journal of Periodontology, 2007; 78(3): 498–503.
29. S. Dabra, K. China, and A. Kaushik, "Salivary enzymes as diagnostic markers for detection of gingival/periodontal disease and their correlation with the severity of the disease," Journal of Indian Society of Periodontology, 2012; 16(3): 358–364.