

## Lactate Dehydrogenase (LDH) Levels in Chronic Periodontitis

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### Abstract

### Original Research Article

**Introduction:** Periodontal disease is a multifactorial disease characterized by chronic inflammatory process that is caused by interaction between virulent microorganisms and host responses, resulting in connective tissue destruction that supports bone. **Aim:** The present study was aimed to assess serum LDH levels in patients of chronic periodontitis and age and sex matched healthy controls. **Materials & Method:** The present study was conducted in the department of Biochemistry in collaboration with the Department of Periodontics, PGIMS, Rohtak. A total of 60 subjects were enrolled in the study, which were divided into two groups. Group I was the case group which included 30 newly diagnosed patients of chronic periodontitis and group II was the control group which included 30 age & sex matched subjects. LDH levels were analyzed by autoanalyser on the same day in both cases and controls. **Results:** In the present study mean serum LDH levels were found to be higher in chronic periodontitis patients as compared to their age and sex matched healthy controls. (426.12 ± 109.25 U/L and 141.8 ± 49.11 U/L respectively). This difference was found to be statistically highly significant (p=0.000). **Conclusion:** Based on the results of our study it was concluded that serum LDH levels were increased in chronic periodontitis. Therefore serum LDH can be considered as feasible and useful biochemical marker in diagnosis of chronic periodontitis

**Keywords:** Periodontitis, LDH, Biomarker, Periodontal disease.

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## INTRODUCTION

Periodontal disease is a multifactorial disease characterized by chronic inflammatory process that is caused by interaction between virulent microorganisms and host responses, resulting in connective tissue destruction that supports bone around the teeth resulting in progressive attachment loss and tooth loss subsequently [1,2]. Periodontitis is usually diagnosed by probing the soft gum tissues, clinical attachment level, and plaque index and by radiographs. Diagnostic procedures play a significant role in providing location, and severity of the periodontal disease which serve as a basis for treatment and management of disease [3]. Traditional diagnostic procedures were sufficient only to assess the disease history and not the ongoing changes in periodontal disease. However, recent advances in diagnostic research enables the identification of periodontal risk with the help of various biochemical markers [4].

Biomarker is used as an indicator of biological state in healthy individuals and periodontal disease. Biochemical marker can detect inflammatory changes associated with the disease process in short period of

time [5]. In oral diagnostics, it has been a great challenge to determine biomarkers for screening, prognosis and evaluating the disease activity and the efficacy of therapy [6].

Lactate Dehydrogenase (LDH) is an enzyme present in cytoplasm of every cell. During the process of glycolysis usually the end product is pyruvate but in anaerobic conditions pyruvate is reduced to lactate by a reversible reaction catalyzed by lactate dehydrogenase. It becomes extracellular after cell necrosis, tissue destruction and cell death caused by ischemia, excess heat and cold, starvation, dehydration, injury, exposure to bacterial toxin, ingestion of some drugs and certain chemicals. It is mostly used in medicine as a diagnostic indicator to assess cell destruction and damage [5, 7, 8].

The present study was aimed to assess serum LDH levels in patients of chronic periodontitis and age and sex matched healthy controls.

## MATERIALS AND METHODS

The present study was conducted in the department of Biochemistry in collaboration with the

Department of Periodontics, PGIMS, Rohtak. A total of 60 subjects were enrolled in the study, which were divided into two groups. Group I was the case group which included 30 newly diagnosed patients of chronic periodontitis and group II was the control group which included 30 age & sex matched subjects. An informed consent was obtained from all patients before enrolling them in the study. Patients were diagnosed with periodontitis according to following criteria: The subjects with at least 20 teeth, and had at least eight sites with PD > 4 mm and attachment level > 2mm. were included in the study. The periodontically healthy subjects had at least 24 natural teeth, no probing depth > 3 mm and no attachment level > 2mm [9]. Subjects with any known systemic diseases, subjects who had used antibiotics during the previous six months, Pregnant and lactating women, subjects who use

tobacco in smoked or smokeless form were excluded from the study.

Blood sample was drawn aseptically from cases and control after taking consent. Serum was separated by centrifugation and LDH levels were analyzed by autoanalyser on the same day. Student T-test was applied to compare cases and controls. Data wee expressed in mean ± standard deviation (SD). p <0.05 considered a significant and p≤0.001 considered as highly significant.

## RESULTS

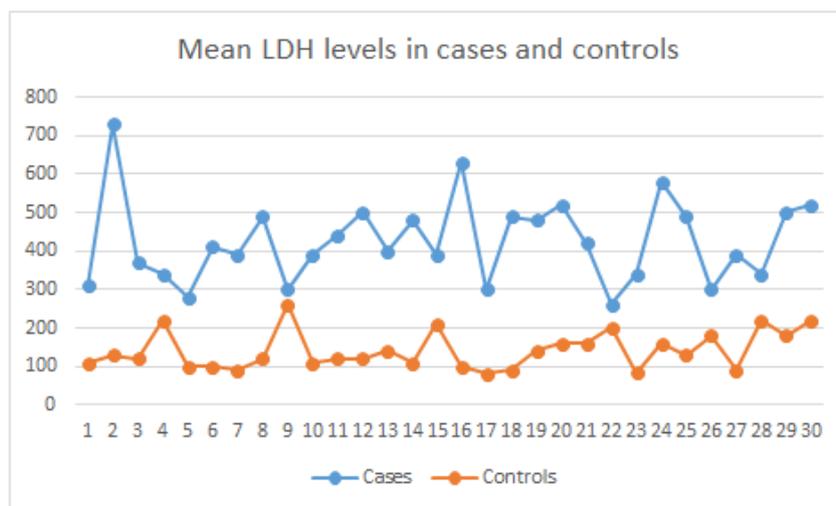
### Comparison of LDH (U/L) levels in Cases and controls

**Table-1: Showing means LDH levels in both the groups**

Parameter	Cases	Control	Significance
Mean LDH (U/L)	426.12 ± 109.25	141.8 ± 49.11	P=0.000

In the present study mean serum LDH levels were found to be higher in chronic periodontitis patients as compared to their age and sex matched healthy

controls. (426.12 ± 109.25 U/L and 141.8±49.11 U/L respectively). This difference was found to be statistically highly significant (p=0.000).



**Fig-1: showing mean LDH levels in both the groups**

## DISCUSSION

Periodontal diseases are mainly diagnosed by clinical and radio graphical parameters but these parameters will provide only limited information about the risk sites for future periodontal connective tissue breakdown which lately results in tooth loss in periodontics individuals. Serum diagnostic biomarkers are most commonly used for the detection of various systemic diseases [10]. Many of this biomarker is used to diagnose, treat and evaluate the course of periodontitis such as LDH and alkaline phosphatase (ALP). These biomarkers usually remains within normal limits in healthy individuals. However in case of periodontitis edema results in cells damage, or

destruction of the cellular membrane occurs which further leads to be increased release of these biomarkers into the gingival crevicular fluid, serum and saliva [11].

In the present study mean serum LDH levels were found to be higher in chronic periodontitis patients as compared to their age and sex matched healthy controls. This difference was found to be statistically highly significant (p=0.000, shown in table-1 and figure 1). The results of our study are in agreement with Kalburgi *et al.* and smith *et al.* who reported that LDH levels increase with increased probing depth in periodontitis patients as compared with healthy individuals having normal probing depth [5, 12].

LDH is an enzyme which plays a remarkable role in the clinical findings of pathologic processes. [13] LDH is present especially in the muscle, liver, myocardium, kidney and erythrocytes. Marked increase of the enzyme activity of LDH is found in myocardial infarction, toxic liver damage or testicular cancer. Moderate increments of LDH were also found in muscle disease, hemolysis and malignant lymphoma [14].

Serum LDH has a widespread distribution in the body. It is released into the peripheral blood after cell death caused by ischemia, excess heat or cold, starvation, dehydration, injury, exposure to bacterial toxins, after ingestion of certain drugs, and from certain chemicals resulting in raised serum LDH levels[6]. This alteration of cellular enzyme levels indicates some abnormality may be due to an altered amount of the enzyme forming tissue, an altered rate of synthesis of the enzyme within the tissue of origin, or an alteration in the permeability of cell membrane caused by pathological disease states [15]. Increased serum LDH levels in chronic periodontitis patients may be due to cellular necrosis seen in periodontal disease.

This increase in LDH levels may be a warning sign even if it was not a statistically significant value. Beck and colleagues have postulated a connection between periodontal disease and atherosclerosis. As such, people suffering from periodontal disease may be at increased risk of atherosclerosis [16].

## CONCLUSION

Based on the results of our study it was concluded that serum LDH levels were increased in chronic periodontitis. Therefore serum LDH can be considered as feasible and useful biochemical marker in diagnosis of chronic periodontitis. Future Studies with larger sample size should be carried out to validate these findings.

## REFERENCES

1. Socranky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. *J periodontol.* 1992;63:322-31
2. Genco RJ. Host response in periodontal diseases; current. *J periodontol.* 1992; 63:338-55.
3. Ozmeric N. Advances in periodontal disease markers. *Clin Chim Acta.* 2004; 343:1-16.
4. Shu L, Guan SM, Fu SM, Guo T, Cao M, Ding Y. Estrogen Modulates Cytokine Expression in human periodontal Liagment cells. *J Dent Res.* 2008;87:142-7
5. Kalburgi NB, Koregol AC, Thomas T, Warad S, Pattanshetti J, Kataria N. Serum lactate dehydrogenase: A cross link between chronic periodontitis and tobacco. 2017;6: 50-2.
6. Havle AA, Suragimath G, Zope SA, Varma SA, Ashwinirani, Hvle KA. Comparison and correlation of lactate dehydrogenase levels of saliva and serum of healthy subject patients, patients with gingivitis and chronic periodontitis. 2017; 9:53190-3.
7. Rutger Persson G, DeRouen TA. Relationship between levels of aspartate aminotransferase in gingival crevicular fluid and gingival inflammation. *J Periodontal Res.* 1990;25: 17-24.
8. Gandolfo S, Pentenero M, Broccoletti R, Pagano M, Carrozzo M, Scully C. Toluidine blue uptake in otentially malignant oral lesion in vivo: clinical and histological assessment. *Oral Oncol.* 2006; 42:89-95.
9. Teles R, Sakellari D, Teles F, Konstantinidis A, Kent R, Socransky S, Haffajee A. Relationships among gingival crevicular fluid biomarkers, clinical parameters of periodontal disease, and the subgingival microbiota. *Journal of periodontology.* 2010 Jan;81(1):89-98.
10. Ganapathi A, Vishnupriya Vand Gayathri R. Study of enzyme biomarkers to evaluate periodontal disease. *J Pharm Sci & Res.* 2016; 8:696-9.
11. Deepika V, Vishnu Priya V, Aroonika Bedre, Harsha L. Salivary AST, ALP and CK Levels in Patients with Periodontitis. *J Pharm Sci & Res.* 2015; 7:341-3.
12. Smith QT, Au GS, Freese PL, Osborn JB, Stoltenberg JL. Five parameters of gingival crevicular fluid from eight surfaces in periodontal health and disease. *J Periodontal Res.* 1992; 27:466-75.
13. De La Pen VA, Dios DP, and Sierra RT. Relationship between lactate dehydrogenase activity in saliva and oral health status. *Arch Oral Biol.* 2007; 52:911-5.
14. Kornberg A, Polliak A. Serum lactate dehydrogenase levels in acute leukemia. Marked elevations in lymphoblastic leukemia. *Blood.* 1980; 56(3):351-5.
15. Merza KS, Alaaraji SB, Abdullah BH. Comparative study on lactate dehydrogenase, alkaline phosphatase and immunoglobulins in serum and saliva of acute leukaemia and oral squamous cell carcinoma patients. *Iraq J Sci.* 2010; 51:262-7.
16. Beck J, Gracia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease an cardiovascular disaease. *J Periodontol.* 1996; 67:1123-37.