

SERUM ALKALINE PHOSPHATASE LEVEL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS RELATION WITH PERIODONTITIS

Asmat Shaheen,¹ Salim Khattak,¹ Aziz Marjan Khattak,¹ Asghar Kamal,¹ S A Jaffari,² Alam Sher³

ABSTRACT

Objective: To study the level of serum alkaline phosphatase in diabetic patients with periodontitis and compare to control group.

Material and Methods: This study was carried out in Pathology Department Gomal Medical College Dera Ismail Khan, from Jan. 2006 to June 2007. Subjects of type-2 diabetes mellitus having periodontitis (group A), diabetics without periodontitis (group B), non-diabetics with periodontitis (group C) and control subjects (non-diabetics and without periodontitis) were included. Patients of both sexes aged between 31 and 70 years were studied. Blood samples were analysed for fasting, post-prandial sugar and serum alkaline phosphatase levels. Depth of periodontal pocket \geq 3mm was labeled as a patient suffering from periodontitis. A WHO recommended probe gently placed to the base of the pocket and the depth of insertion was read. At least six points on each tooth; mesio-buccal, mid-buccal and disto-buccal and the corresponding lingual sites were measured. Patients with other diagnosed diseases affecting ALP levels were excluded.

Results: No significant difference in fasting and post prandial blood sugar level in control and the non-diabetics with periodontitis. ALP showed significant rise in both diabetics and non-diabetics with periodontitis. ALP is raised slightly in diabetics without periodontitis but significantly with periodontitis (groups A & C), however, depth of pockets was very significantly more in patients with periodontitis compared to control and diabetics without periodontitis (group B).

Conclusion: Periodontitis is a debilitating disease leading to significant rise in level of ALP in subjects with type 2 diabetes mellitus as well as in non-diabetics.

Key words: Periodontitis, Type 2 Diabetes Mellitus, Alkaline Phosphatase.

This article may be cited as: Shaheen A, Khattak S, Khattak AM, Kamal A, Jaffari SA, Sher A. Serum alkaline phosphatase level in patients with type 2 diabetes mellitus and its relation with periodontitis. KUST Med J 2009; 1(2): 51-54.

INTRODUCTION

Alkaline phosphatase is hydrolytic enzyme acting optimally at pH 10. Active center of alkaline phosphatase include a serine residue. Magnesium (Mg) and zinc (Zn) ions are also required for minimal activity. Physiological increases are found during bone growth, while patho-

logical increases are largely associated with hepatobiliary and bone diseases¹.

Phosphatases are enzymes that catalyze the splitting of phosphoric acid from monophosphoric esters. Two types are commonly estimated in serum, a mixture of *alkaline phosphatase* with maximum activity at about pH 10 and *acid phosphatase* at pH 5 and 6.

Diabetes mellitus (DM) is a clinical syndrome characterized by abnormal metabolism of carbohydrate, protein and fat resulting in hyperglycemia due to absolute or relative deficiency of insulin ending up in vascular complications leading to retinopathy, neuropathy and nephropathy. It can be divided into two main categories. Insulin Dependent Diabetes Mellitus (IDDM) now labeled as type-1 Diabetes Mellitus and Non-insulin Dependent Diabetes Mellitus (NIDDM) known as type-2 Diabetes Mellitus². Type-2 Diabetes Mellitus constitutes 85-90% of diabetic patients. Uncontrolled diabetes (chronic hyperglycemia) is associated with several long-term complications, related with micro-vascular diseases including retinopathy, nephropathy, neuropathy and macro-vascular diseases such as cardiovascular and cerebrovascular, increased susceptibility to infection; and poor

- 1 KUST Institute of Medical Sciences (KIMS), Kohat, Pakistan.
- 2 Institute of Molecular Biology and Biotechnology, The University of Lahore.
- 3 Biochemistry Department, Gomal Medical College, Dera Ismail Khan.

Address for correspondence

Dr. Asmat Shaheen

Assistant Professor Biochemistry
KUST Institute of Medical Sciences
Kohat, Pakistan
asmatshaheen@yahoo.com
03347217349

Date received: 15 September 2009

Date revised: 20 November 2009

Date accepted: 04 December 2009

wound healing. It has been reported that many diabetics may also exhibit elevated serum alkaline phosphates level⁹.

The increased prevalence and severity of periodontitis is commonly seen in diabetics, the sixth complication of diabetes; in the sequence of retinopathy, nephropathy, neuropathy, macrovascular disease and altered wound healing². In addition to the five classic complications of diabetes, the American Diabetes Association has officially recognized that periodontal disease is common in diabetics and its standards of care include taking a history of current or past dental infections as part of the physician's examination³.

Initially periodontal disease (gingivitis) is seldom painful and causes relatively minor signs. Untreated gingivitis can lead to periodontitis, a serious infection that destroys the soft tissue and bone supporting teeth and eventually may cause tooth loss. Long term periodontitis can lead to more serious problems like heart attack and stroke⁴.

Various enzymes are released from host cells during the initiation and progression of periodontal disease^{5,6}. 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^{7, 8}.

The clinical features of oral cavity are very important for DM like Xerostomia, glossopyrosis and oral candidiasis and impaired wound healing⁹.

Neutrophils as immune competitive cells carry out a protective function of an organ. These are the first cells that migrate into tissues, phagocytate microbes and their complexes and devast injured tissues¹⁰. Periodontal disease is frequently diagnosed in diabetic patients. There is relation between injured periodontal tissues and DM. The injured tissue secrete alkaline phosphatase from the neutrophil that causes destruction of connective tissues and level of ALP activities directly correlate with the intensity of inflammatory process of periodontal tissue¹¹.

The purpose of study was to investigate a possible correlation between raised ALP levels and periodontitis in type 2 diabetics and non-diabetics.

MATERIAL AND METHODS

This study was carried out in Pathology Department Gomal Medical College Dera-Ismael Khan, from Jan 2006 to June 2007.

In this study, there were 96 diabetics type-2 of which 46 were having periodontitis (group A), 40 diabetics without periodontitis (group B), 40 non-diabetics with periodontitis (group C) and 40 control subjects (non diabetics and without periodontitis) age range from 31-70 years.

Male and female patients with Type-2 diabetes aging 31-70 years, fasting blood glucose > 125 mg/dL and post-prandial > 200mg/dL and willing to give consent were included in the study. Patients with other diagnosed diseases affecting ALP levels were excluded. Measurements are expressed as Mean ± SEM. Results were analyzed using chi square test.

Clinical Assessment Of Periodontitis

British periodontal examination (BPE) as recommended by the British Society of Periodontology (BSP) is a rapid screening system, which has been developed to aid the identification of risk patients with a significant periodontal problem. Depth of pocket ≥ 3mm is labeled as a patient suffering from periodontitis.

A special WHO recommended probe, is gently placed to the base of the pocket and the depth of insertion is read. At least six points on each tooth should be examined; mesio buccal, mid buccal and disto buccal and the corresponding lingual sites.

RESULTS

There were 96 patients having type-2 Diabetes mellitus with (group A) or without periodontitis (Group B) of the age range 31 to 70 years. Forty non-diabetics with periodontitis (group C) of sex and age comparison also included. Forty normal subjects of comparable sex and age range included as control group. The figures are given in Table I.

REPRESENTS NUMBER OF SUBJECTS IN DIFFERENT GROUPS AND AVERAGE AGE

Groups	No. of subjects	Average age in years
T2DM with periodontitis (Group A)	56	45.5
T2DM without periodontitis(Group B)	40	45.5
Non-diabetics With peridontits (Group C)	40	45.1
Control (Normal subjects)	40	46.3

Table I

LEVELS OF BLOOD GLUCOSE AND SERUM ALKALINE PHOSPHATASE (ALP) AND THE DEPTH OF PERIODONTAL POCKETS IN THE CONTROL SUBJECTS AND PATIENTS WITH THE AGE RANGE OF 31-70 YEARS.

S. No.	Subjects/Patients	Blood Glucose fasting level (mg/100ml)	Blood Glucose level post prandial (mg/100ml)	Alkaline Phosphatase levels (UL)	Depth of periodontal pockets (mm)
1.	Control Subjects	85.2 ± 0.827	136.9 ± 2.282	146 ± 2.216	0.00
2.	Diabetics with Periodontitis (Group A)	171.1 ± 15.497	294.7 ± 18.226	253.9†* ± 10.006	4.86†*§ ± 0.042
3.	Diabetics without Periodontitis (Group B)	142.2* ± 12.313	274.5* ± 22.153	149.5 ± 0.636	0.00
4.	Non-diabetics with Periodontitis (Group C)	89.5 ± 1.746	133.1 ± 1.561	224.2†* ± 4.983	3.98†*§ ± 0.048

Each value is the mean ± SEM of observed values.

* P < 0.05 When values in different groups compared with control .

† P < 0.05 When compared Group A with Group C.

§ P < 0.01 When compared Group A and Group C with control.

Table II

The mean values of blood glucose (fasting and postprandial), serum alkaline (ALP) and the depths of periodontal pocket in (mm) in diabetics with periodontitis, diabetics without periodontitis, non-diabetics with periodontitis and control subjects within age range of 31-70 years are given in Table II. No significant difference in fasting and post-prandial blood glucose has been observed between control subjects and non-diabetics with periodontitis (Group C). However ALP showed the significance (P < 0.05) rise in both diabetics and non-diabetics with periodontitis Group A & C) as compared with controls. Comparing groups of diabetics and non-diabetics both having periodontitis (Group A & C) with control, it can be seen that there is very significant (P < 0.01) increase in ALP and depth of periodontal pockets.

DISCUSSION

A number of studies have shown raised serum ALP levels in various physiological and pathological conditions e.g. during bone growth physiologically while pathological increases are associated with hepatobiliary and bone diseases¹. Diabetes mellitus is a metabolic disorder arising from insulin insufficiency², which is associated with altered activity of various enzymes e.g. ALP, SGPT and SGOT etc.¹². Besides the microvascular and macro vascular complications in diabetes mellitus a compromised immune state is also a condition that increase the susceptibility of diabetes to different infec-

tion, particularly including opportunist micro-organisms such as constituting oral micro flora¹³.

ALP showed a significant rise in both diabetic and non-diabetic patients with periodontitis as compared to control. Diabetic patients even in the absence of periodontitis showed a slight increase in mean values of serum ALP. Comparing diabetics (Group A) and non-diabetics (Group C) both having periodontitis, raised blood glucose level in diabetic patients and change in medium make the individual susceptible to infection due to depressed immunity¹⁴. It can be seen that significant increase of ALP and depth of pockets in diabetics with periodontitis (Group A) and non-diabetics with periodontitis (Group C)⁸.

The broad spectrum of pathogen is defeated by the individuals when immunity is intact.¹⁵ Raised value of serum ALP and diabetic patients has been reported by Grossi, 1998¹⁶; Iwamoto et al, 2001¹⁷; Siddiqui et al, 2005.¹² Raised value of serum ALP in our population with periodontitis, particularly when they are diabetics is in agreement with a number of studies¹⁸.

CONCLUSION

Patients with periodontitis have an increased level of serum ALP and increased depth of periodontal pockets and these were a little worse in diabetics having periodontitis.

REFERENCES

1. Moss DW. Release of membrane bound enzymes from cells and the generation of isoforms. *Clin Chem Acta* 1994; 226: 131-42.
2. Loe H. The sixth complication of diabetes mellitus. *J Diabetes Care* 1993; 16: 476 - 80.
3. Enlow DH. Physiologic tooth movement and alveolar remodeling. In Enlow DH, ed. *Facial Growth*. Philadelphia; Saunders, 1999; 130 -148.
4. Mochida Y, Duarte WR, Ranzawa H, Paschalis EP, Yamauchi M, Decorin modulates matrix mineralization in vitro. *Biochem Biophys Res Commun* 2003; 305: 6-9.
5. Renggli HH: Phagocytosis and killing by cervicular neutrophils. In Lehner T (ed): *Periodontal disease and diabetes mellitus*. New York, Grune and Stratton, 1977.
6. Palmer J.P, Culloch M C. Prediction and prevention of IDDM. *Diabetes* 1982; 40: 943-7.
7. De Bernard B. Glycoproteins in the local mechanisms of calcification. *J Clin Orthop* 1982; 162: 233-44.
8. Safkon-Seppala B, Ainaimo J. Periodontal condition in insulin dependent diabetes mellitus. *J Clinical periodontal* 1992; 19: 24-9.
9. Benson L. Arthodontic treatment considerations in patients with diabetes mellitus. *Am J Orthod Dentofac Arthrop* 2003; 1: 74-8.
10. Biasi D, Carletto A, Dell Angola C. Neutrophil migration, oxidative metabolism adhesion in elderly and young subjects. *Inflammation* 1999; 20: 673.
11. Aainamo J, Lahtinen A, Uitto V. Rapid periodontal destruction in adult human with poorly controlled diabetes. *J Clin Periodontal* 1990; 17: 22-8.
12. Siddiqui SA, Cheema AM, Waheed M. Study of serum insulin, liver profile and protein levels of insulin resistant type-2 diabetics in Pakistan population. *Pak J Biochem Mol Biol* 2005; 38(3-4); 92-7.
13. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non insulin dependent diabetes mellitus. *J Periodontal* 1991; 62: 123-31.
14. Bulkacz J. Enzymatic activities in gingival fluid with special emphasis on phospholipases. *J Western Soc Periodont* 2001; 36: 145.
15. William R, Mahan C. Periodontal disease and diabetes in young adults. *JAMA* 1960; 172: 776-8.
16. Grossi SG, Genco RJ, Machtei E. Periodontal disease and diabetes mellitus: A two way relationship. *J Ann Periodontal* 1988; 3: 51-61.
17. Iwamoto Y, Nishimura F, Nakagawa M, Sugimoto H, Shikata K, Makino H, et al: The effect of antimicrobial periodontal treatment on circulating tumor necrosis factor α and glycated hemoglobin level in patients with type 2 diabetes. *J Periodontal* 2001; 72: 774-8.
18. Armitag G. Diagnostic tests for periodontal diseases. *J Curr Opinion Dent* 1992; 2: 53.

CONFLICT OF INTEREST

Authors declare no conflict of interest

KUST Medical Journal**Online submission of articles**VISIT <http://kmj.kust.edu.pk/>

REGISTER > LOG IN > HOME > USER > AUTHOR >
 SUBMISSIONS > NEW SUBMISSION (FOLLOW-5
 STEPS) > START > ENTER METADATA > UPLOAD
 SUBMISSION > UPLOAD SUPPLEMENTARY FILES >
 CONFIRMATION