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Original Research

Assessment of Alkaline Phosphatase Level in Tobacco Users

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ABSTRACT:

Background: Cigarette smoking is known to be one of the major causes of various health disorders. The present study was conducted to determine alkaline phosphatase (ALP) level in tobacco users. **Materials & Methods:** The present study was conducted on 60 patients of both genders. Patients were divided into 4 groups of 15 each depending upon oral lesion and habit of tobacco use. In all subjects, venous blood was obtained to assess alkaline phosphatase level. **Results:** Out of 60 subjects, males were 40 and females were 20. The mean serum alkaline phosphatase level in group I was 17.3 IU/L, in group II was 8.5 IU/L, in group II was 5.6 IU/L and in group IV was 61.5 IU/L. The difference found to be significant (P< 0.05). **Conclusion:** Authors found that ALP level was higher in subjects with intraoral lesion and with the habit of smoking/chewing tobacco. ALP may be considered as a reliable noninvasive biomarker in monitoring potentially malignant disorders.

Key words: Alkaline phosphatase, potentially malignant disorders, Smoking.

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INTRODUCTION

Saliva is an oral fluid that has been used as a diagnostic tool in medicine and dentistry. The source of the specimen that can be used for salivary markers are whole saliva, gingival crevicular fluid (GCF) and plaque. Among these, enzymes released from the host can be easily obtained within the oral cavity either from GCF or from the whole saliva.[1] Several enzymes evaluated for the early diagnosis of periodontal disease are lactate dehydrogenase, alkaline phosphatase (ALP), acid phosphatase, aspartate aminotransferase and alanine aminotransferase. Sampling technique for GCF collection is a time-consuming process and is a difficult procedure.[2]

Oral potentially malignant disorders (OPMDs), a terminology suggested by the World Health Organization in 2007 for premalignant lesions and conditions, has been reported with a high-risk percentage of malignant transformation to oral squamous cell carcinoma (OSCC). OSCC accounts for over 30% of all malignancies in the Indian population. Although many etiologic factors have been proposed, tobacco product is a well-established etiology for the development of OPMD and OSCC.[3]

Cigarette smoking is known to be one of the major causes of various health disorders. These toxic components can predispose to different systemic disorders, such as cardiac diseases, cancers, precancerous lesions and pulmonary disorders. Saliva is the first body fluid to encounter cigarette smoke. The salivary antioxidant system plays a very important role in the anti-carcinogenic capacity of saliva and includes various enzymes and molecules, such as uric acid, peroxidase system and phosphatases.[4] The present study was conducted to determine alkaline phosphatase (ALP) level in tobacco users.

MATERIALS & METHODS

The present study comprised of 60 patients of both genders. All patients were informed regarding the study and written consent was obtained. Ethical clearance for the study was taken from institutional ethical committee. Data related to patients such as name, age, gender etc. was recorded. Patients were divided into 4 groups of 15

each. Group I had subjects without the habit of smoking or chewing tobacco and without any oral lesion, group II had subjects with the habit of chewing tobacco and without any intraoral lesion, group III had subjects with the habit of smoking and without any lesion intraoral lesions and group IV subjects with intraoral lesion and with the habit of smoking/chewing tobacco. In all subjects, venous blood was obtained to assess alkaline phosphatase level. Results thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 60		
Gender	**Males**	**Females**
Number	40	20

Table I, graph I shows that out of 60 subjects, males were 40 and females were 20.

Graph I Distribution of patients

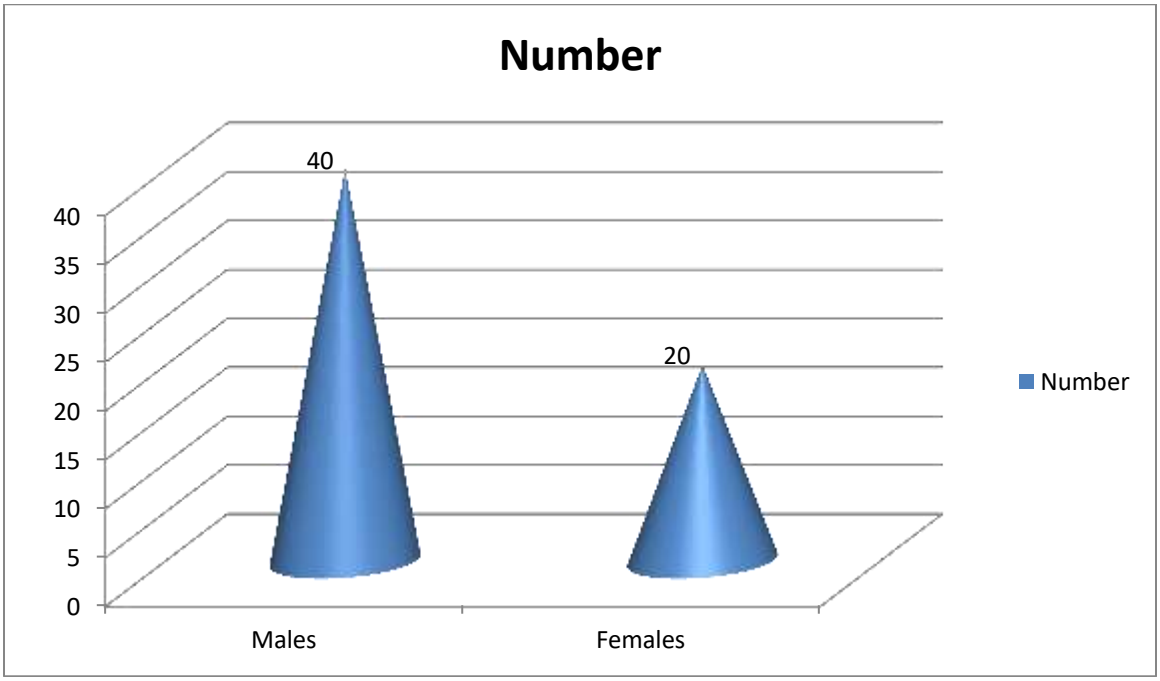
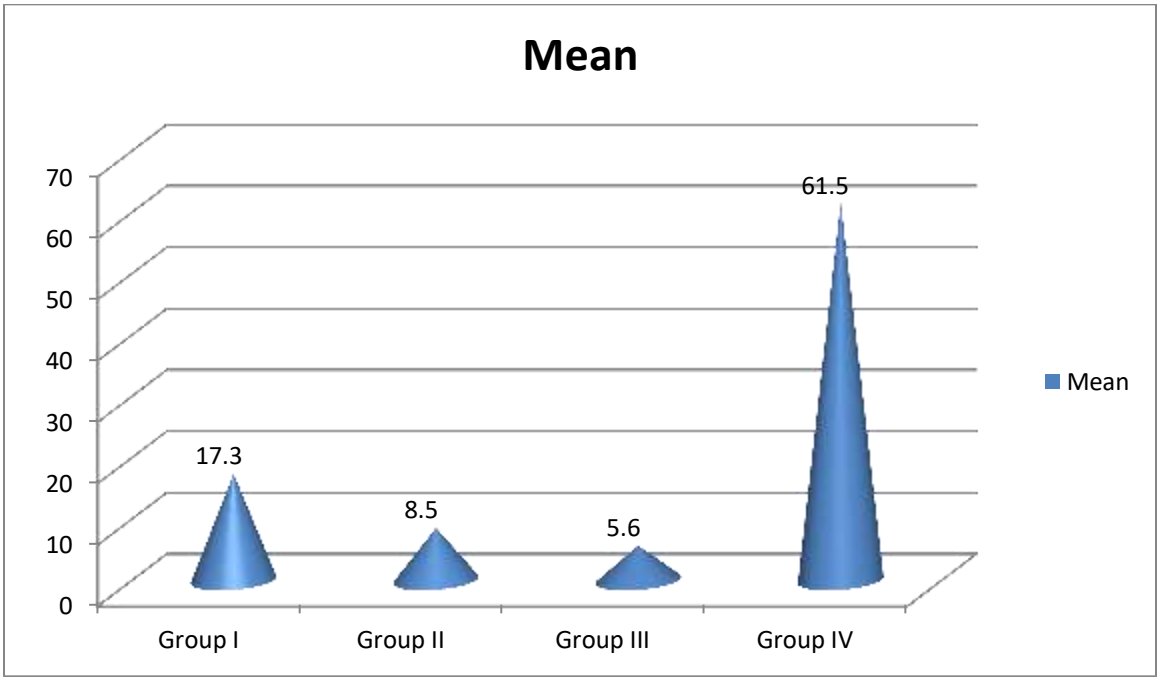


Table II Comparison of salivary alkaline phosphatase level in groups

Groups	Mean	SD	P value
Group I	17.3	4.5	0.001
Group II	8.5	2.1	
Group III	5.6	1.7	
Group IV	61.5	32.7	

Table II, graph II shows that mean serum alkaline phosphatase level in group I was 17.3 IU/L, in group II was 8.5 IU/L, in group II was 5.6 IU/L and in group IV was 61.5 IU/L. The difference found to be significant (P< 0.05).

Graph II salivary alkaline phosphatase level in groups



DISCUSSION

ALP is a membrane-bound glycoprotein found on most cell membranes in the body and physiologically occurs during bone formation in developmental stages.[5] It is produced by many cells within the periodontal environment, the principal source being PMNs leukocytes, bacterial fibroblast and osteoblast activity which is disturbed due to diabetes, smoking, etc., pathologically. ALP is one of the potentially powerful markers of periodontal disease activity and ALP levels increases in periodontal diseases.[6]

Toxic products due to smoking levels depend on the balance between their rates of production and their rates of clearance by the endogenous antioxidant systems, including superoxide dismutase (SOD), catalase (CAT), the glutathione (GSH) redox cycling enzymes, GSH-Px, reductase and GSH itself. One of the principal reactive oxygen species produced in aerobic organisms is O_2^-, which is highly cytotoxic. With the cytotoxicity of this oxidant, exposure to cigarette smoke results in increased levels of antioxidant enzymes, such as CAT, copper/zinc SOD, Px and GSH-Px.[7] The present study was conducted to determine alkaline phosphatase (ALP) level in tobacco users.

In present study, out of 60 subjects, males were 40 and females were 20. The mean serum alkaline phosphatase level in group I was 17.3 IU/L, in group II was 8.5 IU/L, in group II was 5.6 IU/L and in group IV was 61.5 IU/L. The difference found to be significant ($P< 0.05$).

Prakash et al[8] determined the levels of S-ALP in diagnosing potentially malignant conditions and debilitating diseases in early stages of inflammation and altered cellular metabolism. The study groups include: Group A - 10 smokers who are diabetic. Group B - 10 smokers who are nondiabetic. Group C - 10 nonsmokers who are diabetic. Group D - 10 nonsmokers and non-diabetic as control. Unstimulated saliva samples are collected and run in auto-analyzer with ALP enzyme reagent to analyze ALP levels. Results were statistically significant with increased activity of ALP levels in saliva from Group A when compared to Group D. The results are Group A > Group B > Group C > Group D. The results also revealed significant raise in levels of ALP levels in saliva from smokers when compared to diabetes thus explaining adverse effects of smoking on ALP level.

Dhivyalakshmi et al[9] study comprised of 42 individuals, categorized into four groups with/without tobacco usage habit and with/without lesion. 5 ml of unstimulated saliva sample was collected and S-ALP was estimated in the supernatant by using kinetic photometric method in an automatic analyzer. The mean S-ALP was 18.00 IU/L for normal individuals without tobacco usage, 4.60 IU/L for smokers without lesion, 7.50 IU/L for tobacco chewers without any lesion and 64.90 IU/L for individuals with OPMD. The mean difference between the groups was statistically significant ($P < 0.001$) using Kruskal–Wallis' ANOVA. No statistically significant difference ($P > 0.05$) was obtained in the S-ALP levels between tobacco users and nonusers and between smokers and tobacco chewers, using Mann–Whitney U-test. S-ALP levels in individuals with OPMD were

statistically significantly higher ($P < 0.001$) than those without lesions, with or without tobacco usage habit.

It is observed that the higher reactive O_2 is converted to H_2O_2 by SOD. CAT, Px, or GSH-Px can, in turn, convert H2 O_2 to molecular oxygen and water. Under physiologic conditions, these systems tend to maintain a stable state called redox homeostasis. The imbalance between the formation of free oxygen radicals and inactivation of these species by antioxidant is capable of causing damage to various cellular and extracellular constituents. At the same time, S-ALP evaluation which is altered due to antioxidant imbalances can act as alarm in smokers and diabetics. Cigarette causes destruction of vascular collagen.[10]

CONCLUSION

Authors found that ALP level was higher in subjects with intraoral lesion and with the habit of smoking/chewing tobacco. ALP may be considered as a reliable noninvasive biomarker in monitoring potentially malignant disorders.

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