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Periodontitis as a causative agent for systemic disease of pancreas

Sweeti Jain¹, Veena Sharma², M.K.Gupta³, Mukul Tailang⁴ and A.K.Pathak¹

¹Department of Pharmacy, B.U. Bhopal M.P, 462026, India.

²Department of Bioscience, Banasthali Vidyapeeth, Jaipur, India.

³People's Dental Academy Bhopal, 462037, India.

⁴People's Institute of Pharmacy And Research Centre – 462037, India.

Abstract

In recent years it has been studied that periodontal diseases have a strong correlation with systemic health. The purpose of the present study is to correlate the periodontitis with the pancreatic health. Two hundred sixty subjects were selected for this study and divided in control and chronic periodontitis. Amylase, lipase, glucose, cholesterol, sodium, potassium and calcium level were measured in serum of all subjects. Significant changes were observed in amylase, glucose, cholesterol and calcium levels in periodontitis subjects.

Keywords: Periodontitis; Pancreatic health; Amylase; Cholesterol

INTRODUCTION

The periodontal disease is confirmed by a group of illness affecting the gums and dental support structures (1). Periodontal health can be considered to be a state of balance when the bacterial population coexists with the host and no irreparable damage occurs to either the bacteria or the host tissues. Disruption of this balance causes alteration in both the host and biofilm bacteria and results ultimately in the destruction of the connective tissues of the periodontium. The pancreas is one of the important organ of the digestive tract, which play important role in metabolism via their hormones like insulin, glucagon or their enzymes like trypsin, chymotrypsin. Any changes in pancreatic function can lead to systemic damage in major extents. Several studies suggested an association between periodontitis and systemic health (2,3).

MATERIAL AND METHODS

One hundred two hundred sixty subjects of both sexes; aged 25-60 were selected from People's Dental College and Hospital Bhopal, India. Subjects were divided into 4 groups: Group A (control group), Group B, Group C and Group D (Test groups)

Group A consisted of 50 healthy subjects without any systemic manifestation, Group B consisted of 70 subjects with early chronic periodontitis, Group C consisted of 70 subjects with moderate chronic periodontitis and Group D consisted of subjects with severe chronic periodontitis. Smokers and persons with any other systemic disease were excluded from study. Periodontal diseases were diagnosed by dentist on the basis of clinical

parameters. Blood sample was collected by venipuncture from each participant, in a sterile test tube without anticoagulant. Serum was isolated by centrifugation for 30 minutes at 4°C and 2000 rpm. The amylase was determined by the method of Somogi (4), lipase by the method of Cherry and Crandal (5), glucose by the method of Kurt (6), Cholesterol by the method of Zak (7), and electrolytes by flame photometer.

RESULTS

There were no significant changes observed in serum lipase activity and serum sodium concentration in test groups compared to control. The amylase activity of early periodontitis subjects is not associated significantly compared to control, but in the subjects with moderate and severe periodontitis it is increased significantly at the level of $p < 0.05$ and $p < 0.01$, respectively. A linear increment is reported in the mean glucose concentration in test groups according to severity of disease compared to control. Like glucose, cholesterol concentration is also increased significantly in test groups with the disease severity. The subjects with severe periodontitis exhibited significantly high ($p < 0.05$) serum potassium value compared to control, but no significant association is observed in group B and C. Calcium value decreased significantly ($p < 0.05$) in subjects with early, moderate periodontitis compared to control and in severe periodontitis it is decreased at the level of $p < 0.01$.

DISCUSSION AND CONCLUSION

To the best of our knowledge, it is the first study showing a relation between periodontitis with pancreas function test and serum electrolytes level. Although serum Lipase activity and sodium are not affected by the periodontitis in present study, but a deviation is observed in amylase, glucose and lipase activity due to periodontitis. Simultaneous changes in calcium and potassium level are recorded in periodontitis subjects. These findings suggest a fluctuation in pancreas function, due to periodontitis and therefore positive relation between periodontitis and pancreas functioning state. It has been

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*Corresponding Author

A.K.Pathak
Department of Pharmacy, B.U. Bhopal M.P, 462026, India.

Email: akpathak.bub@gmail.com

proved from previous studies that periodontitis pathogens and their products found in systemic circulation; simultaneously stimulate the production of cytokines (8). These bacteria, bacterial products along with cytokines can cause pancreas tissue damage and appeared in the form of impaired pancreas function. On the basis of the study it is stated that good oral health is important not only to prevent oral

disease but also to maintain good general health.

It is concluded that pancreas is an organ which regulate the metabolic process in other body organs. Any impairment in pancreatic function can influence other organs of the body and initialize a disease condition.

Table 1. Parameters for Pancreas Function Test in Different study Groups

Serum Markers	Group A Mean \pm SE	Group B Mean \pm SE	Group C Mean \pm SE	Group D Mean \pm SE	P value ANOVA
Lipase (IU/L)	198.56 \pm 6.58	208.21 \pm 5.39*	211.99 \pm 5.57*	213.43 \pm 5.91*	>0.05
Amylase (IU/L)	95.06 \pm 2.58	99.61 \pm 2.16*	101.56 \pm 1.93**	103.86 \pm 2.10***	>0.05
Glucose (mg/dl)	99.68 \pm 1.73	106.29 \pm 2.32**	111 \pm 2.58***	112.17 \pm 2.73****	<0.01
Cholesterol (mg/dl)	197.42 \pm 3.03	207.31 \pm 3.33**	208.84 \pm 2.97***	214.07 \pm 3.40****	<0.01
Sodium (meq/l)	141.5 \pm 0.48	142.08 \pm 0.33*	142.45 \pm 0.39*	142.72 \pm 0.39*	>0.05
Potassium (meq/l)	4.49 \pm 0.07	4.60 \pm 0.07*	4.59 \pm 0.07*	4.71 \pm 0.07**	>0.05
Calcium(mg/dl)	9.84 \pm 0.07	9.65 \pm 0.06**	9.62 \pm 0.06**	9.56 \pm 0.06***	<0.05

*P value calculated by t test compared to control * >0.05, **<0.05, ***< 0.01, ****<0.001, SE = Standard Error

REFERENCES

- [1] Bascones Martinez A, Figuero Ruiz E. 2005. Periodontal disease as bacterial infection. *Av Periodontol Implantol*, 17, 3: 11-118.
- [2] Beck JD, Garcia RI, Heiss G, Vokonas PS, Offenbacher S. 1996. Periodontal disease and cardiovascular disease. *J Periodontol* 67:1123-1137.
- [3] Pussinen PJ, Alfthan G, Rissanen H, Reunanen A, Asikainen S, Knekt P. 2004. Antibodies to periodontal pathogens and stroke risk. *Stroke*, 35:2020-2023.
- [4] Somogyi M. 1960. Modification of the two methods for the assay of amylase. *Clin Chem*, 6:23.
- [5] Kurt M Duwoski. 1962. An O-toluidine method for body fluid glucose determination. *Clinical Chemistry*, 8:215-235.
- [6] Cherry IS, Crandall LA. 1932. The specificity of pancreatic amylase: its appearance in blood after pancreatic injury. *Am J Physiol*, 100:266.
- [7] Zak B. 1957. Simple rapid microtechnique for serum total cholesterol. *Amer J Clin Path*, 27:583-588.
- [8] Scannapieco FA. 2004. Periodontal inflammation: from gingivitis to systemic disease? *Compend Cont Sduc Dent*, 25(7) (Suppl 1): 16-25.