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Original Research

Antioxidant status in type II diabetics and non diabetics: A biochemical study

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***ABSTRACT*:**
Introduction: Diabetes mellitus is a heterogeneous group of metabolic disease showing features of hyperglycemia which results from disorders in carbohydrate, fat and protein metabolism. Free radicals are produced which induce lipid peroxidation which acts as an indicator for oxidative stress in the body. Defensive system in the body consists of antioxidant enzymes like reduced glutathione (GSH) and superoxide dismutase (SOD) which help in scavenging free radicals. **Aims and objective**: To estimate and correlate the levels of GSH and SOD in saliva and serum of both diabetics and non-diabetics individuals. **Materials and method:** Unstimulated saliva and venous blood samples were obtained after 12 hoursofovernight fast and the samples were transported to the laboratory. Supernatants of the centrifuged samples were used for the determination of GSH and SOD. **Results:** The antioxidant enzymes i.e. GSH revealed a statistically significant decrease in their values among the diabetic groups. The levels of SOD in the serum and saliva of diabetic individuals indicated a significant increase in their values when compared with the serum and saliva of control individuals.There was a strong significant positive correlation between serum GSH and salivary GSH in diabetic as well as in control group. **Conclusion:** Saliva and serum can be used as a diagnostic tool for the estimation of GSH and SOD.
Key words: Diabetes mellitus, antioxidant enzymes, saliva, serum, reduced glutathione

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INTRODUCTION:
The term diabetes has its origin from Greek language and literally means "siphon" and was introduced in pathology by 'Aretaeus of Cappadocia'. The word mellitus means 'honey' which was added to the name diabetes by 'Cullen'.[1] WHO defined diabetes mellitus is a heterogeneous group of metabolic disease showing features of hyperglycemia which results from disorder in carbohydrate, fat and protein metabolism.[2] Hyperglycemia may result from decreased insulin secretion, reduced glucose uptake by the body and increased amount of glucose production. The various risk factors associated with diabetes mellitus are family history of diabetes, obesity, smoking, hypertension, infertility, previous gestational diabetes.[3] Diabetes mellitus can be classified as – Type I diabetes mellitus and Type II diabetes mellitus. Type I diabetes mellitus is an autoimmune disease which results from destruction of beta cells in Islets of Langerhans caused by immune effector cells against endogenous beta-cell antigens and give rise to absolute insulin deficiency. Type II diabetes mellitus can be defined as a heterogeneous multifactorial disease which results from either due to insulin resistance or impaired insulin secretion and leads to relative insulin deficiency. Approximately 5-10% of cases have type I diabetes and occurs mostly in children and young adults whereas type II diabetic mellitus accounts for approximately 90-95% of cases and mostly associated with adults. The signs and symptoms of diabetes mellitus include polyuria, polyphagia, nocturia, polydipsia, fatigue, unexplained weight loss, increased infections, numbness, leg cramps, blurred vision and impotence.[4, 5] World health organization demonstrated that in the year 2000, the total number of people affected in India was 32 million but the rate is increasing day by day due to population growth, obesity, aging and development. As per International diabetes federation that 40.9 million people was affected in India in the year 2000 and this rate increases to 69.9 by the year 2030. [6, 7]
The immune system of an individual works in anexactway for the sustenance of the normal equilibrium, thus aiding in achieving a diseasefree state. The immune

system can function overprotective at various intervals thus results in increased emission of free radicals, which then produce oxidative stress and lipid peroxidation.[8] The rise in the lipid peroxidation and decline in the antioxidant defense may appear early in noninsulin-dependent diabetes mellitus before the appearance of secondary complications and could play an important role in the initiation and progression of acute and chronic diabetic complications like diabetic ketoacidosis, non-ketotic hyperosmolar state, nephropathy, neuropathy and dermatological, gastrointestinal, genitourinary and micro and macrovascular complications.[9]

Lipid peroxidation occurs due to reactive oxygen species (ROS) and causes significant changes in the cell membrane. It has been associatedwith the pathogenesis of various degenerative diseases like atherosclerosis, carcinogenesis, diabetes mellitus and aging. Lipid peroxidation forms a well-established mechanism of cellular injury.[10-12]

Aerobic cells have established their own defense system called as antioxidant protection system so as to control the influx of ROS. This system includes enzymatic and non-enzymatic components which then neutralize the free radicals. The antioxidant enzymes that are produced endogenously are superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase and reduced glutathione (GSH) as well as the free radicalscavenging substances such as Vitamins E, C and carotenoids are exogenous in nature.[13] The aim of the present study was to estimate and correlate the levels of reduced glutathione (GSH) and superoxide dismutase (SOD) in saliva and serum of both diabetics and non-diabetics.

MATERIALS AND METHOD:

A descriptive cross-sectional study was conducted in a private set up on 50 subjects including 25 diabetic (Group I) and 25 non-diabetics subjects (Group II). All these subjects were briefly informed about the test procedure before sample collection and an informed consent was obtained from all subjects. The sample collection was done in the morning between 8:00 am and 9:00 am to avoid diurnal variation. Blood and unstimulated saliva samples were collected after 12 hours of overnight fast. All the subjects included in this study were assessed for the fasting blood glucose, erythrocyte sedimentation rate (ESR), body mass index (BMI) and postprandial blood sugar.

Inclusion criteria

1. Diabetics: Subjects with type II diabetes mellitus confirmed by fasting blood sugar, under medication (oral hypoglycemic drugs and insulin) in the age group of 35–65 years.
2. Controls: Normal healthy nondiabetic subjects in the age group of 35–65 years.

Exclusion criteria

1. Diabetics: Individuals with uncontrolled diabetes, habits like smoking and alcoholism, systemic diseases such as coronary artery disease and renal diseases, patients under any medication other than oral hypoglycemic drugs and insulin and subjects with a history of any illness for the past 6 months.
2. Controls: Individuals with any chronic systemic diseases, habits, patients under any medication other than oral hypoglycemic drugs and insulin and subjects with a history of any illness for the past 6 months.

Sample collection: 3ml of unstimulated saliva was collected by drooling the saliva into the vial and 3 ml of venous blood was collected under aseptic condition from each subjectinto a vial. Samples taken were transported in ice bags at a temperature range of 0°C–4°C to the laboratory. Saliva and serum samples were cold centrifuged at 3000 rpm for 5 min. The supernatant was aspirated and was stored at −20°C until analyzed. The clear supernatant sample was used for the biochemical analysis of GSH and SOD with the help of a“spectrophotometer.”

RESULTS:

The data was analysed by using statistical software (SPSS version 19.0). Mean and standard deviation were calculated for cases and controls. A probability value (p) of ≤0.05 was considered to be statistically significant .

In the present study, the levels of serum and salivary GSH and SOD were evaluated both in diabetic and control groups (Table 1).

Table1: Biochemical parameters in serum and saliva of diabetics and control group GSH: Glutathione, SOD: Superoxide dismutase,SD: Standard deviation

Parameters	Mean ± SD		P-value
	Diabetics (n=25)	Control (n=25)	
Serum GSH (mg/dl)	12.50±2.75	24.04±6.14	<0.001
Serum SOD (U/ml)	1.42±0.4	1.12±0.4	<0.001
Salivary GSH (µmol/l)	2.09±0.2	2.07±0.2	<0.001
Salivary SOD (U/ml)	1.62±0.4	1.42±0.4	<0.001

Table 2: Correlation coefficient (*r*) between glutathione (GSH) and superoxide dismutase (SOD) in serum and saliva of both diabetics and nondiabetics

Correlation between	Serum GSH (mg/dl)		Serum SOD (U/ml)	
	r	p	R	p
Salivary GSH (mg/dl)	0.965***	<0.001		
Salivary SOD (U/ml)			0.063	0.550

The mean serum levels of GSH (mg/dl) in diabetic and control groups were 12.50±2.75 and 24.04±6.14 respectively. The p-value was found to be statistically significant. The mean salivary levels of GSH (µmol/l) in diabetic and control groups were 2.09±0.2 and 2.07±0.2 respectively. The p-value was found to be statistically significant. The antioxidant enzymes i.e. GSH revealed a statistically significant decrease in their values among the diabetic groups. The mean serum levels of SOD (U/ml) in diabetic and control groups were 1.42±0.4 and 1.12±0.4 respectively. The p-value was found to be statistically significant. The mean salivary levels of SOD (U/ml) in diabetic and control groups were 1.62±0.4 and 1.42±0.4respectively. The p-value was found to be statistically significant. The values of SOD increased significantly among the diabetic groups. There was a strong correlation of SOD in serum and saliva, but it was statistically significant (Table 2).

DISCUSSION:

Type II diabetes mellitus is related with several metabolic derangements which can cause secondary pathophysiological variations in multiple organ systems. This can impose a heavy burden of morbidity and mortality from micro and macro-vascular complications. [14] Diabetes can cause persistent hyperglycemia often accompanied with other features like glycosuria, polydypsia and polyuria which then can cause increased production of free radicals in all tissues from glucose auto oxidation and protein glycosylation.[15]In the present study, we determined the antioxidant enzymes i.e. GSH and SOD in the serum and saliva of 25 subjects with type II diabetes mellitus and 22 subjects of healthy individuals and correlated the serum values with the saliva values. All the subjects in our study with type II diabetic revealed a significant decrease in antioxidant GSH both in the serum and saliva as compared with the serum and saliva of control individuals. This finding was in agreement with the study done by Al-Rawi[10]and Nair et al[16]. They determined that oxidative consume some naturally occurring local antioxidants such as GSH which may evident as reduced levels in the serum and saliva of diabetic patients.

In the present study, the antioxidant SOD in the serum and saliva of diabetic individuals indicated a significant increase in their values when compared with the serum and saliva of control individuals. These results were in accordance with the studies done by Al-Rawi[10], Nair et al [16] and Padalkar[17]. They showed that an increase in the antioxidant SOD might be due to the body's mechanism to raise the antioxidant defense so as to counteract the increasing oxidative stress.

In the present study, a correlation of serum GSH and SOD with their corresponding salivary values were determined. There was a strong significant positive correlation between serum GSH and salivary GSH in diabetic as well as in control group. In this study, a weak positive correlation between serum SOD and salivary SOD as well as the p-value was not statistically significant. All the results were in agreement with the study done by Nair et al. [16.]

CONCLUSION:

Saliva can be used as a "diagnostic tool" in an area of research as it can offers various advantages over serum. The collection process of saliva is noninvasive and can be performed easily as well as cost effective. In the present study, we assessed the antioxidant status by measuring GSH and SOD in the serum and saliva of diabetics and non-diabetics individuals. Saliva and serum can be used as a diagnostic tool for the estimation of GSH and SOD.

REFERENCES

1. Rahman K. Diabetes mellitus and its oral complications: A brief review. Pakis Oral Dent J 2006; 26(1):97-100.
2. Harsh Mohan. The endocrine system. Textbook ofpathology 5th ed. New Delhi: Medical publisher 2005;842-845.
3. Matthews DC. The relationship between diabetes andperiodontal disease. J Can Dent Assoc 2002; 68(3):161-164.
4. Kumar V, Abbas AK, Fantaso N and Aster JC. Theendocrine system. Robbins and Cotran Pathologic basisof disease 8th ed. Elsevier 2013; 1134-1137.
5. Seino Y et al. Report of committee on the classificationand diagnostic criteria of diabetic mellitus. DiabetolInt2010; 1:2-20.
6. Mohan V, Sandeep S, Deepa R, Shah B and VargheseC. Epidemiology of type 2 diabetes: Indian scenario.Indian J Med Res 2007; 125:217-230.
7. Mohsin SF, Ahmed SA, Fawwad A and Basit A.Prevalence of oral mucosal alterations in type 2 diabetesmellitus patients attending a diabetic center. Pak J MedSci 2014; 30(4):716-719.
8. Arora R, Vig PA, Arora S. Lipid peroxidation: A possible marker for diabetes. J Diabetes Metab 2013; S11:1-6.
9. Maritim AC, Sanders RA, Watkins JB. Diabetes, oxidative stress, and antioxidants: A review. J BiochemMolToxicol 2003; 17:24-38.
10. Al-Rawi NH. Oxidative stress, antioxidant status and lipid profile in the saliva of type 2 diabetics. DiabVasc Dis Res 2011; 8:22-28.
11. Rahman K. Studies on free radicals, antioxidants, and co-factors. ClinInterv Aging 2007; 2: 219-236.
12. Halliwell B, Chirico S. Lipid peroxidation: Its mechanism, measurement, and significance. Am J ClinNutr 1993; 57:715S-724S.
13. Percival M. Antioxidants. ClinNutr Insights 1998; 31:1-3.
14. Moussa SA, Youssef AA. Oxidative stress in diabetes mellitus. Rom JBiophys 2008;18:225-36.
15. Grodsky GM, Anderson CE, Coleman DL, Craighead JE, Gerritsen GC, Hansen CTet al. Metabolic and underlying causes of diabetes mellitus. Diabetes 1982; 31:45-53.
16. Nair A and Nair BJ. Comparative analysis of theoxidative stress and antioxidant status in type II diabetics and nondiabetics:A biochemical study. J Oral MaxillofacPathol 2017;21:394-401.
17. Padalkar RK, Shinde AV, Patil SM. Lipid profile, serum malondialdehyde,superoxide dismutase in chronic kidney diseases and type 2 diabetesmellitus. Biomed Res 2012;23:207-10.