

CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.3525180

Available online at: <u>http://www.iajps.com</u>

Research Article

ANALYSIS OF ROLE OF ANTIOXIDANTS AND OXIDATIVE STRESS IN THE DEVELOPMENT OF DIABETES TYPE II IN PAKISTAN

Aamir Suhail¹, Ghulam Murtaza², Muhammad Asad Shahzad³

¹Government Rural Dispensary Niwani District Bhakkar

²Bahawalpur Victoria Hospital

³Basic Health Unit Matrian District Lodhran

Abstract:

Introduction: Oxidative stress is caused by an unfavorable balance between reactive oxygen species (ROS) and antioxidant defenses. ROS are generated during normal cellular metabolism, as a result of the influence of various environmental factors, as well as during pathological processes. Objectives: The main aim of our study is to find the role of antioxidants and measure the level of oxidative stress and their role in the cause of diabetes type II in Pakistani population. Methodology: This cross sectional study was conducted at Bahawalpur Victoria Hospital during January 2019 to July 2019. For this purpose we selected the patients who was suffering from a common disease diabetes. This study is based on the local population of Pakistan, which shows the stress level in Pakistani environment. Results: According to analysis of data level of antioxidant and oxidative stress is increasing in diabetic patients because cell become destroyed. GSH is important non-enzymatic antioxidant which helps in scavenging of free radical mechanism. Conclusion: It is concluded that level of antioxidants in our body plays an important role. It is obvious from the presented data that a relation exists between hyperglycaemia, oxidative stress, cellular and endothelial dysfunction.

Corresponding author:

Aamir Suhail, Government Rural Dispensary Niwani District Bhakkar



Please cite this article in press Aamir Suhail et al., Analysis Of Role Of Antioxidants And Oxidative Stress In The Development Of Diabetes Type Ii In Pakistan., Indo Am. J. P. Sci, 2019; 06(10).

INTRODUCTION:

Oxidative stress is caused by an unfavorable balance between reactive oxygen species (ROS) and antioxidant defenses. ROS are generated during normal cellular metabolism, as a result of the influence of various environmental factors, as well as during pathological processes. Reactive oxygen species play an important role in the pathogenesis of cancer.¹ Oxidative stress caused by increased free radical generation and/or decreased antioxidant level in the target cells and tissues has been suggested to play an important role in carcinogenesis.² Free radicals are capable of altering all major classes of biomolecules, such as lipids, nucleic acids and proteins, with changes in their structure and function. Prime targets of free radicals are the polyunsaturated fatty acids in cell membranes and their interaction results in lipid peroxidation. The levels of free radical molecules are controlled by various cellular defense mechanisms, consisting of enzymatic (catalase, glutathione peroxidase, superoxide dismutase) and non-enzymatic (vit. E, vit. C, glutathione) components.³

ROS can be produced endogenously or exogenously. In vivo free radicals are created during normal aerobic respiration, by commencement of phagocytosing cells, in peroxisomes where fatty acids are degraded, and by auto-oxidation of various molecules. The mitochondria plays very important role and it is a major physiologic source of reactive oxygen species (ROS), which can be generated during mitochondrial respiration.⁴ Super oxide radicals, formed by minor side reactions of the mitochondrial electron transport chain or by an NADH-independent enzyme can be converted to H_2O_2 and to a powerful oxidant, the hydroxyl radical. Oxidative stress in organisms leads to the peroxidation of all major biomolecules, such as DNA, proteins and lipids. The most widely used method to find oxidative stress is to determine lipid peroxidation with the Thiobarbituric acid reactive substances (TBARS) method. Among these targets, the peroxidation of lipids is basically damaging because the formation of lipid peroxidation product leads to spread of free radical reactions. The general process of lipid peroxidation consists of three stages: initiation, propagation and termination.⁵

Objectives

The main aim of our study is to find the role of antioxidants and measure the level of oxidative stress and their role in the cause of diabetes.

MATERIAL AND METHODS:

This cross sectional study was conducted at Bahawalpur Victoria Hospital during January 2019 to July 2019. For this purpose we selected the patients who was suffering from a common disease diabetes. This study is based on the local population of Pakistan, which shows the stress level in Pakistani environment.

Data collection

5.0 ml blood sample was taken from vein. Blood was further processed for the estimation of GSH, Catalases, SOD, MDA, Neuraminidase and Sialic acid. Commercially available enzymatic kits of Randox were used. Blood was centrifuged at 4000 rpm for 10 minutes and serum was separated. Blood samples will be collected into EDTA tubes from fasting proteins. The blood will be centrifuged and indomethacin and butylated hydroxytoluene will be added into the plasma samples before they will be stored at -80°C until analysis. The sample were processed and analyzed for the estimation of SOD, GSH, CATALASES, MDA, NO, neuraminidase and sialic acid levels.

Statistical analysis

The collected data were analyzed using SPSS software (version 20). The results are presented as a mean with 95% confidence interval limits or standard deviations. The significant value for P < .05 was accepted as statistically significant.

RESULTS:

According to analysis of data level of antioxidant and oxidative stress is increasing in diabetic patients because cell become destroyed. GSH is important nonenzymatic antioxidant which helps in scavenging of free radical mechanism. According to data the levels of GSH become decreases in diabetic patients. The data pertaining in the table shows that levels of sialic acid become increases in patients. The level becomes increases in all cases. As the value in this case is 3.48 ± 0.65 . According to our data MDA is considered to be an important antioxidant and serum stress biomarker in case of diabetic patients.

Variable	CONTROL (moles/ml)	(moles/ml) (n=100)
		Diabetic patients
SOD	0.22	25.074
SOD	0.32	3.5±0./4
MDA	2.35	3.6±0.82
Catalases	4.16	0.00 ± 0.00
SOD	0.326	3.27±0.16
Sialic acid	0.37	1.05 ± 0.08
GSH	8.26	3.48±0.65

Table 01: Level of anti-oxidants in control and diabetic patients

Means±SD

DISCUSSION:

Reactive oxygen species (ROS) cause oxidation of DNA, proteins and lipids, and induce carcinogenesis. Some studies have reported high lipid peroxidation levels become high in human colorectal cancer tissue and gastric cancer tissue. The major aldehyde products of lipid peroxidation are malondial-dehyde (MDA) and 4hydroxynonenal. MDA is mutagenic in mammalian cells and carcinogenic.6

Peroxidation of lipids can disturb the assembly of the membrane, causing changes in fluidity and permeability, alterations of ion transport and inhibition of metabolic processes. Injure to mitochondria induced by lipid peroxidation can direct to further ROS generation.⁷ Catalase is a common enzyme found in nearly all living organisms which are exposed to oxygen, where it functions to catalyze the decomposition of hydrogen peroxide to water and oxygen. Catalase has one of the highest turnover numbers of all enzymes; one molecule of catalase can convert millions of molecules of hydrogen peroxide to water and oxygen per second.⁸

Superoxide is one of the main reactive oxygen species in the cell and as such, super oxide dismutase (SOD) serves a key antioxidant role. The physiological importance of SODs is explained by the severe pathologies evident in mice genetically engineered to lack these enzymes. In mammals there are several types of SODs, which differ with respect to their location in the cell and the metal ion they require for their function. For example, a copperzinc SOD is present in the fluid filling the cell (i.e., the cytosol) and in the space between two membranes surrounding the mitochondria.⁹ Furthermore, a manganese-containing SOD is present in the mitochondrial interior. Both of these

enzymes are critical for prevention of ROS-induced toxicity.

Glutathione (GSH) is a molecule which contains three peptide linkages. It is an antioxidant, and it helps to protect the cells from ROSs and free radical damages. It contain three amino acids; cysteine, glutamic acid and glycine.¹⁰

CONCLUSION:

It is concluded that level of antioxidants in our body plays an important role. It is obvious from the presented data that a relation exists between hyperglycaemia, oxidative stress, cellular and endothelial dysfunction.

REFERENCES:

- Baynes J.W., Thorpe S.R. Role of Oxidative Stress in Diabetic Complications: a new perspective on an old paradigm. Diabetes 1999 Jan; 48(1): 1 – 9.
- 2. Bownlee M. Glycation and diabetic complications. Diabetes. 1994; 43: 836- 841.
- Cosentino F., Luscher T.F. Endothelial dysfunction in diabetes mellitus. J Cardiovasc Pharmacol. 1998; 32:S54-S61.
- Guigliano D., Ceriello A., Paolisso G. Oxidative stress and diabetic vascular complications. Diabetes care. 1996; 19: 257-266.
- Kesavulu M.M., Giri R., Kameswara Rao B., Apparao C. Lipid peroxidation and antioxidant enzyme levels in type 2 diabetics with microvascular complications. Diabetes Metab. 2000 Nov; 26: 387-92.
- King G.L., Shiba T., Olivier J., Induchi T., Brussell S.E. Cellular and molecular abnormalities in the vascular endothelium of diabetes mellitus. Ann Rev. Med. 1994; 45: 179-188.

IAJPS 2019, 06 (10), 13987-13990

- Koya D, King GL: Protein kinase C activation and the development of diabetic complications. Diabetes, 1998; 47:859–866.
- Ruderman N.B., Williamson J.R., Brownlee M: Glucose and diabetic vascular disease. FASEB J, 1992; 6:2905–2914.
- 9. Miata T., Kurokava K. Advanced glycation and lipoxidation end products. J Am Soc Nephrol, 2000; 11:1744-1752.
- 10. Ho E, Bray TM: Antioxidants, NFkappaB: activation, and diabetogenesis. Proc Soc Exp Biol Med, 1999;222:205–213.