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ROLE OF OXIDATIVE STRESS IN THE DEVELOPMENT OF DIABETES MELLITUS

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Abstract:
Objectives: *The main objective of our study is to find the level of oxidative stress and their role in the cause of diabetes.* ***Material and Methods:*** *This cross-sectional study was conducted at Lahore General Hospital, Lahore during June 2019 to January 2020. This study is based on the local population of Pakistan, which shows the stress level in Pakistani environment. 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.* ***Results:*** *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. 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.* ***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.*

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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. 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 [1]. 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 [2]. Prime targets of free radicals are the polyunsaturated fatty acids in cell membranes and their interaction results in lipid peroxidation [3]. 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 [4].

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 [5]. 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 [6].

Objectives of the study

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

MATERIAL AND METHODS:

This cross-sectional study was conducted at Lahore General Hospital, Lahore during June 2019 to January 2020. 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.

The collected data were analyzed using SPSS software (version 17). 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 non-enzymatic 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.

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

Variable	**CONTROL (moles/ml)**	**(moles/ml) (n=100) Diabetic patients**
SOD	0.32	3.5±0.74
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

DISCUSSION:

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 4-hydroxynonenal. MDA is mutagenic in mammalian cells and carcinogenic [9].

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 [10]. 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 [11].

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:

1. Baynes JW. Role of oxidative stress in development of complications in diabetes. Diabetes. 1991; 40: 405-412.
2. 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.
3. Bownlee M. Glycation and diabetic complications. Diabetes. 1994; 43: 836- 841.
4. Cosentino F., Luscher T.F. Endothelial dysfunction in diabetes mellitus. J Cardiovasc Pharmacol. 1998; 32:S54- S61.
5. Guigliano D., Ceriello A., Paolisso G. Oxidative stress and diabetic vascular complications. Diabetes care. 1996; 19: 257-266.
6. 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.
7. 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.
8. Koya D, King GL: Protein kinase C activation and the development of diabetic complications. Diabetes, 1998; 47:859– 866.
9. Ruderman N.B., Williamson J.R., Brownlee M: Glucose and diabetic vascular disease. FASEB J, 1992; 6:2905– 2914.
10. Miata T., Kurokava K. Advanced glycation and lipoxidation end products. J Am Soc Nephrol, 2000; 11:1744-1752.
11. Ho E, Bray TM: Antioxidants, NFkappaB: activation, and diabetogenesis. Proc Soc Exp Biol Med, 1999;222:205–213.