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Oxidic Degradation of Lipids in Patients with Type II Diabetes

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SUMMARY: Lipid peroxide-mediated damage has been observed in the development of type 2 diabetes mellitus. The aim of this study was the measurement of oxidic degradation of lipids in diabetic patients. The degree of lipid peroxidation was determined by the reaction of thiobarbituric acid with malondialdehyde, a secondary product of lipid peroxidation. The absorbance of clear supernatant was measured against reference blank at 530 nm. Plasma MDA levels in diabetics were observed to be statistically significantly higher than in control group ($p < 0.05$).

INTRODUCTION

Diabetes is a clinical entity whose incidence and prevalence are increasing. Globally, 1 in 11 people have diabetes, and it is estimated that by 2030 this ratio will have risen to 1 in 10 (1). Type 2 diabetes affects almost 90% of people with diabetes and is very often associated with obesity. Central (or abdominal) obesity leads to insulin resistance, hyperinsulinemia, and impaired glucose tolerance. When pancreatic β -cells, which normally produce insulin, begin to malfunction and become deficient, glucose levels rise above the normal range and type 2 diabetes occurs. The symptoms that a person with type 2 diabetes may have are mainly related to elevated blood glucose levels (hyperglycemia), and include the classic symptoms of overeating and polydipsia, pruritus, increased incidence of infections. Factors that may increase risk of type 2 diabetes include: weight, fat distribution, family history, blood lipid levels, age,

inactivity, prediabetes, Pregnancy-related risks, Polycystic ovary syndrome (2,3,4). Oxidative stress represents a disturbance of the balance between its production of reactive oxygen species (ROS) and ability of a biological system to inactivate these toxic molecules and repair the damage they cause. Active forms of oxygen harm every cell component, including proteins, lipids and DNA. Oxidative stress causes a variety of damage to the body; however, active oxygen radicals are also involved in processes important for body function like: Defense immunity, Cell proliferation, Metabolism (especially H_2O_2), Apoptosis, Muscle contraction, Angiogenesis, Enzyme substrates. Insulin signaling appears to involve H_2O_2 as part of the mechanism for reversible inactivation of certain tyrosine protein phosphatases, while at the same time tyrosine protein kinases are activated via insulin receptor (5). Lipid peroxidation is one of the most commonly reported indices of oxidative stress and has been implicated as a contributing factor in a range of degenerative diseases, including diabetes, cardiovascular disease, Parkinson's disease, Alzheimer's disease, and psychiatric disorders, including schizophrenia (6).

AIM

The aim of this study was the measurement of oxidic degradation of lipids in diabetic patients.

MATERIALS AND METHODS

A total of 160 venous blood specimens from 108 diabetes patients ($65,5 \pm 13$ age) and 52 healthy

volunteers as controls ($55,5 \pm 18$ age) in Athens were collected in an EDTA containing vacutainer tube. Oxidative stress in the cellular environment results in formation of highly active and unstable lipid peroxides by polyunsaturated fatty acids. The product of the breakdown of these unstable molecules is malondialdehyde. Malondialdehyde can be determined by the reaction with thiobarbituric acid (TBA). Thus, TBA Reactive Substances are expressed as equivalents malondialdehyde MDA, which form a compound with TBA with ratio of malondialdehyde to TBA 1/2.

EXPERIMENTAL PROCEDURE

Add 100 μ L of plasma (for samples) or distilled water (for blank) to Falcon test tubes (15 ml). Add 500 μ L of 35% TCA and 500 μ L of Tris-HCl and stir. Incubate for 10 min at room temperature. Add 1 mL $Na_2SO_4(2M)$ – TBA (55mM) and incubate at 95 ° C for 45 min at a water bath. Transfer the Falcon test tubes to the ice and let them cool for 5 min. Add 1 mL of 70% TCA and stir. Transfer 1 mL to Eppendorf's tubes and centrifuge at 11200 g at 25 ° C for 3 min. Pipette 900 μ L of the supernatant into a cuvette and measure absorption at 530 nm (7).

RESULTS

All diabetics had plasma glucose levels of 124-304 mg / dL. Plasma MDA levels in diabetics were observed to be statistically significantly higher than in control group ($p < 0.05$), (Fig.1).

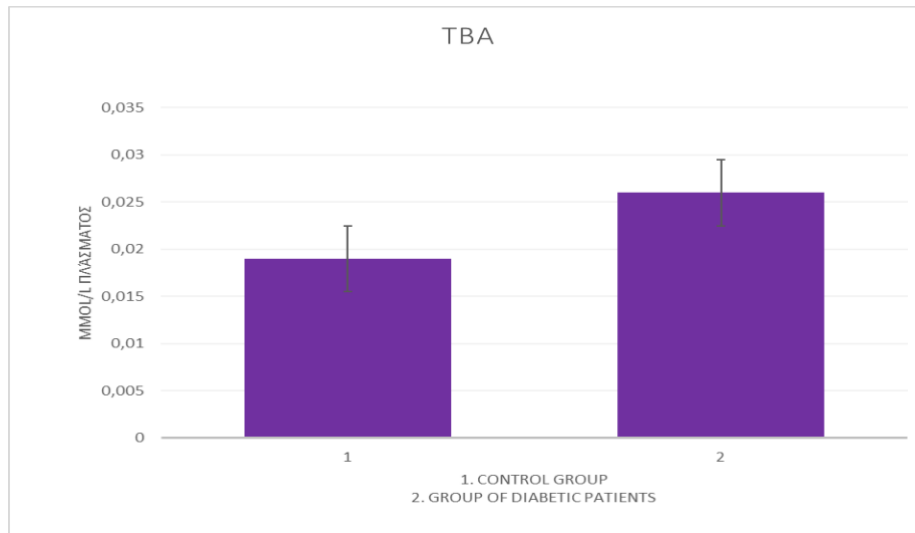


Figure 1: MDA levels in plasma of diabetic patients.

CONCLUSIONS

Lipid peroxide-mediated damage has been observed in the development of type 2 diabetes mellitus. Insulin secretion is also closely associated with lipoxygenase-derived peroxides. Low levels of lipoxygenase peroxides stimulate the secretion of insulin, but when the concentration of endogenous peroxides increases, it may initiate uncontrolled lipid peroxidation leading to cellular infiltration and islet cell damage (8). Significant differences in MDA levels were found between the whole diabetic group and healthy subjects ($p < 0.05$). However, this research will be continued by measuring MDA levels with two chromatographic assays [HPLC-diode array detection (DAD)-fluoro and LC/MS-DAD] for detecting level of lipid peroxidation in clinical samples, and especially in plasma of diabetics.

Conflicts of Interest: The author declares no conflicts of interest regarding the publication of this paper.

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