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Evaluation of Blood Glucose levels and analysis of Malondialdehyde in Diabetic Individuals with Cataracts

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Abstract

Oxidative stress in diabetes can lead to difficulties due to the overproduction of precursors to reactive oxygen radicals and the impaired efficacy of inhibitory scavenger systems. Diabetes mellitus can cause persistent consequences, including senile diabetic cataracts. This study examined NIDDM (Type-2) patients with or without cataracts. Diabetics experience higher levels of oxidative stress, as measured by malondialdehyde (MDA), compared to normal individuals (181.04 nmol/l). Diabetics with cataracts have significantly higher levels of oxidative stress (399.12nmol/l) than diabetics without complications.
The present study attempted to assess serum MDA levels as a measure of free.

Keywords: Diabetes mellitus, Cataract, Malondialdehyde

INTRODUCTION

Diabetics are more likely to develop cataracts at a younger age and a faster rate (Brownlee 1981, Albert 1982). The polyol route involves the enzyme aldose reductase reducing glucose to sorbitol, which has received significant attention.
Diabetes mellitus can cause frequent hyperglycaemia, leading to the buildup of sorbitol. Sorbitol, a tissue toxin, has been linked to several conditions including retinopathy, neuropathy, cataracts, nephropathy, and aortic disease (Brownlee 1981, Albert 1982).

Diabetes is linked to two forms of cataracts.

1. A senile cataract.

2. A true diabetic cataract: This condition is also known as a "snowflake cataract" or "snow-storm cataract".

This rare illness typically affects young adults and is caused by excessive osmotic hydration of the lens. (Comprehensive Ophthalmology, 4th edition, pg. 185). The term "sugar cataract" refers to cataracts caused by galactosemia and juvenile diabetes. This also includes diabetic cataracts in adults with diabetes.

Lipid peroxidation is a chain reaction that generates free radicals and produces malonaldehyde, a stable product. The reaction can be triggered by a free radical (x·), light, or metal ions.

Malondialdehyde, along with ethane from the terminal two carbons of ω 3 fatty acids and pentane from the terminal five carbons of ω 6 fatty acids, are used to assess lipid peroxidation. Oxygen radicals can damage biological molecules, including

1. nucleic acids.

2. Proteins and free amino acids.

3. Lipids and lipoproteins.

4. Carbohydrates

5. Connective tissue and macromolecules.

Diabetic patients' red blood cell membranes produce more Malondialdehyde (MDA), a sign of lipid peroxidation (Nagasaki Y, Fufiis, Kaneko T, 1989). Diabetics have higher amounts of MDA in their plasma than non-diabetics (Nishigaki L, 1981; Altomare E, 1992). Lipid hydroperoxides produce aldehydes like Malondialdehyde, which can indicate oxidative stress. Oxidative damage to lipids has been linked to coronary artery disease and age-related degenerative diseases.

Materials and Methods

Diabetic patients were separated into two groups: those with problems such as cataracts and those without. 75 participants showed oxidative stress as evaluated by MDA, a lipid peroxidation product. Serum MDA levels in patients were compared to healthy volunteers. The study was conducted at outpatient clinics for ophthalmology and internal medicine. The criteria for selecting test and control groups are as follows.

The study includes diabetic participants aged 40 to 70, regardless of gender, who have developed diabetic cataract complications.

Blood samples were collected while fasting to estimate serum malondialdehyde and glucose levels.

Blood glucose levels were analyzed on clinical Biochemistry auto analyzers and Malondialdehyde levels in serum can be estimated using the TBRAS technique.

Statistical analysis: The data thus generated was analysed Statistically using the student 't' test to compare the mean of two groups. ANOVA for comparison of mean in more than two groups. Pearson's coefficient of correlation was used to calculate the correlation between different parameters. $P < 0.05$ was considered statistically significant.

RESULTS

A comparison of 75 people's serum MDA (nmol/l) and fasting blood sugar (mg/dl) levels is observed. Thirty-five of them are diabetics, twenty-five of whom have diabetic cataracts, and twenty-five of whom do not. Of them, twenty-five are normal patients. Using the one-way analysis of variance technique, the data are statistically examined, and the findings are verified. Ten male and fifteen female subjects make up the descriptive data for the fasting blood sugar and MDA levels for 25 normal subjects. Between 80 and 90 mg/dl is the range of blood sugar during fasting. The mean and standard deviation for serum MDA (nmol/l) and fasting blood sugar (mg/dl) are 181.04 ± 22.118 and 86.12± 3.644, respectively. Male and female serum MDA levels range from 147 to 214 nmol/l, which is within the normal range.

describes fasting blood sugar (mg/dl) and MDA for the three sex categories. The fasting blood sugar (mg/dl) of 25 diabetics without cataracts ranged between 129 and 160 mg/dl. The mean and SD for FBS (mg/dl) are 146.76 ± 8.579.

The serum MDA (nmol/l) levels for the same group range from 196 to 507. Serum MDA (nmol) has a mean and standard deviation of 370.80 ± 81.961. The fasting blood sugar levels of 25 diabetic patients with cataracts ranged from 150 to 172 mg/dl, which is above the normal range. The mean and SD for FBS are 161.56 ± 6.602. The serum MDA (nmol/l) levels in this group range from 213 to 540. The mean and SD for MDA (nmol/l) are 399.12±90.719.

DISCUSSION:

The TBARS method measures malondialdehyde, a sign of lipid peroxidation. Our study confirms previous findings (Peuchant E, 1997; Akkus I, 1996; Losada, 1996; Santini SA, 1996; Griesmacher A, 1995; Armstrong D, 1992; Nishigakil, 1981) that diabetes patients have significantly higher levels of serum MDA than healthy individuals. Diabetes is associated with increased oxidative stress and higher levels of Serum MDA compared to non-diabetics.

Normal persons have an average serum MDA level of 181.04 nmol/l, while diabetics without complications have 370.80 nmol/l and diabetic cataract patients have 399.12 nmol/l. The data analysis showed that diabetic cataract patients ($P < 0.05$) and diabetics without problems ($P < 0.05$) had considerably higher serum MDA levels than normal persons. Hyperglycemia has been linked to higher lipid peroxidation and lower antioxidant levels (Auge N, 1995; Ferrari R, 1991).

In studies focusing on a limited number of individuals, antioxidants may not reflect the real antioxidant status of patients. In general antioxidants enzymes such as SOD, CAT, and GPX have been reported to be increased decreased, or unaltered in various tissues of diabetics with wide variation from one tissue to another. These discrepancies may depend on variations in enzyme activity over time. Compensatory increase in enzyme activity to face raised oxidative stress, as well as the type of tissue under examination. (Uzel N, 1987; Wolff SP, 1993; Yeh HC, 1987).

Combining antioxidants improves the antioxidant state and prevents diabetes consequences like cataracts. Diabetes mellitus can cause persistent consequences, including senile diabetic cataracts. Sorbitol is typically found in the lens of the eyes. In diabetes mellitus, increased glucose levels lead to higher sorbitol concentrations in the lens. This damages the tissue and causes cataracts. Elevated sorbitol levels have been linked to diabetes-related neuropathy, cataracts, and retinopathy. Hyperglycaemia accelerates the synthesis of sorbitol, leading to early lens cataract development.

CONCLUSION:

Diabetic patients have much higher levels of oxidative stress (measured by Serum MDA, a lipid peroxidation product) compared to healthy persons. Additionally, diabetic patients with cataracts have significantly higher levels of oxidative stress than those without problems. Lipid peroxidation may potentially contribute to cataract formation. The study found that diabetes patients with and without cataracts have significantly higher serum MDA levels than normal persons.

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