ORIGINAL ARTICLE

Association between oxidative stress index and serum lipid levels in healthy young adults

Kenan Ahmet Turkdogan,[1] Orhan Akpinar,[2] Mustafa Karabacak,[3] Hatice Akpinar,[4] Figen Tunali Turkdogan,[5] Oguz Karahan[6]

Abstract

Objectives: To investigate the relationship between lipid levels and oxidative stress index in healthy young adults.

Methods: The study was camed out at the Department of Emergency Service, Faculty of Medicine, Cumhuriyet University, Sivas, Turkey, between January 2011 and July 2012. A total of 100 healthy adult volunteers were enrolled in the study. Venous blood samples (10 ml) were collected from all individuals, and serum lipid parameters, total antioxidant capacity and total oxidative levels were studied. SPSS 15 was used for statistical analysis.

Results: Overall, there were 84 (84%) males and 16 (16%) females. The mean age fo the male population was 30±3 years, while that of the females was 31±3 years. Overall age ranged from 25 to 35 years. A statistically significant correlation was found between the oxidative stress index and serum cholesterol ($p<0.001$; $r=0.596$), triglyceride ($p<0.001$; $r=0.476$) and low-density lipoprotein levels ($p<0.001$; $r=0.318$). However, no significant correlation was found between oxidative stress index and serum high-density lipoprotein levels ($p=0.564$; $r=0.058$).

Conclusion: The results showed that even at an early age, there is a direct linear correlation between oxidative stress and serum lipid levels.

Keywords: Oxidative stress index, Hyperlipidaemia, Young adult. (JPMA 64: 379; 2014)

Introduction

Under physiological conditions, there is a balance between free radical generation and antioxidant protective defense system. With the shift of this balance towards excessive generation of free radicals, the structure of cell organelles and membrane lipids and proteins are destroyed, intracellular enzymes are inactivated, Deoxyribonucleic acid (DNA) damage occurs, mitochondrial aerobic respiration is blocked, lytic enzymes are activated, loss of intracellular K+ is increased, vascular permeability is decreased, the extracellular connective tissue components are destroyed, and platelet aggregation and migration of phagocytes to the tissues are increased. All these functional and structural changes in the organism are referred to as oxidative stress, which can lead to diseases such as cancer and atherosclerosis, as well as many diseases and physiological conditions can lead to increased oxidative stress.[1]

The fatty tissue, which was only thought to be the reservoir of energy and fat-soluble vitamins in the past, has proven to be a paracrine, autocrine, and even endocrine organ today.[2] Adipocytes and the molecules called adipokine or adipocytokine that are released from surrounding connective tissue have been shown to induce signals that lead to chronic inflammation and increased oxidative stress. In particular, some adipocytokines have been suggested to cause disease processes, including hypertension, insulin resistance, diabetes, and, particularly, atherosclerosis.[3]

In this study, we aimed at investigating the relationship between oxidative stress and serum lipid levels in healthy young adults.

Subjects and Methods

The study was carried out at the Department of Emergency Service, Faculty of Medicine, Cumhuriyet University, Sivas, Turkey, between January 2011 and July 2012. A total of 100 healthy young adult volunteers were enrolled in the study. Those who had some additional disease, consumed cigarettes or alcohol, did heavy exercise, and who had a family history of diseases such as coronary artery disease (CAD), hypertension (HTN), malignancy and diabetes mellitus (DM) were excluded from the study. Venous blood samples (10 ml) were collected from all individuals, and serum lipid parameters, total antioxidant capacity (TAC) and total oxidative levels (TOL) were studied.

Peripheral venous blood samples were drawn from all the

[1]Department of Emergency Medicine, Bezmialem University Medical School, Istanbul, [2]Department of Emergency Medicine, [3]Department of Cardiology, [5]Department of Radiology, Isparta State Hospital, [4]Department of Anestesiology and Reanimation, Faculty of Dentistry, Suleyman Demirel University, Isparta, [6]Department of Cardiovascular Surgery, Dicle University Medical School, Diyarbakir.

Correspondence: Kenan Ahmet Turkdogan. Email: drturkdogan@gmail.com

subjects in the ethylenediaminetetraacetic (EDTA)-plasma tubes. The remaining blood was centrifuged at 4,000 rpm for 4 minutes for plasma separation. Plasma samples were stored at -80°C until analyses for total antioxidant status (TAS), total oxidant status (TOS) and levels of triglyceride (TG), total cholesterol (TC), low density lipoprotein cholesterol, and high density lipoprotein (HDL) cholesterol. Plasma levels of TG, TC, LDL and HDL were measured on an automated analyser (Abbott Aeroset, Abbott Diagnostics, Abbott Park, IL, USA) using commercial kits (Abbott).

The TAS levels of the sera were determined using an automated measurement method based on bleaching of the characteristic colour of a more s 2.2'-azino-bis [3-ethylbenz-thiazoline-6-sulfonic acid (ABTS)] radical cation caused by antioxidants.[4] The results are expressed in mmol Trolox equivalents/L.

The TOS levels of the sera were determined using a novel automated measurement method.[5] Oxidants present in the sample oxidize the ferrous ion-o-dianisidine complexes into ferric ions. The oxidation reaction is enhanced by glycerol molecules that are abundantly present in the reaction medium. The ferric ions form a coloured complex with xylenol orange in an acidic medium. Therefore, the colour intensity, measured spectrophotometrically, is related to the total number of oxidant molecules present in the sample. The assay is calibrated with hydrogen peroxide and the results are expressed in terms of micromolar hydrogen peroxide equivalent per liter (μmol H2O2 equiv./L).

Measurement of oxidative stress index (OSI) is calculated by the following formula:

OSI: TOS μmol H2O2 Equiv/L / TAK mmol trolox Equiv/L x 100

Parametric data was expressed as mean ± standard deviation, and categorical data as percentages. SPSS 15.0 was used to perform statistical procedures. To compare continuous variables cinsiyete göre, Mann-Whitney U test was used as appropriate. Correlations were evaluated either via Pearson or Spearman correlation tests. A p value of 0.05 was considered statistically significant.

Results

Of the volunteers, 84 (84%) were males with a mean age of 30.1±3.3 years and 16 (16%) were females with a mean age of 31.4 ± 3.2 years. The overall mean age of study population was 30.1±3.3 years (range: 25-35 years). Lipid levels were similar in both genders except for total cholesterol. TC levels tended to be higher in the female gender than the male gender (160±38 vs 174±24; p=0.06).

Table-1: Laboratory characteristics.

	Male n: 84	Female n: 16	P value
TC	160±38	174±24	0.06
LDL	140±51	133±44	0.74
TG	110±29	113±28	0.85
HDL	32±5	34±5	0.17
TOS (μmol H_2O_2 Eq/l)	4.19 (1.64-14.7)	4.58 (1.49-7.12)	0.20
TAC (mmolTroloxEq/l)	1.41 (0.98-1.89)	1.35 (1.04-1.59)	0.29
OSI (AU)	3.06 (1.16-9.4)	3.43 (1.2-5.35)	0.24

HDL: High density lipoprotein. LDL: Low density lipoprotein. OSI: Oxidative stress index. TAC: Total antioxidant capacity. TOS: Total oxidant status. TG: Triglyseride. TC: Total cholesterol.
Lipid levels are expressed as mg/dl.

Table-2: Correlation analyses between OSI and serum lipid levels.

	TC	LDL	TG	HDL
OSI (AU)	r=0.596 p<0.001	r=0.318 p=0.001	r=0.476 p<0.001	r=0.058 p=0.564

HDL: High density lipoprotein. LDL: Low density lipoprotein. OSI: Oxidative stress index. TG: Triglyseride. TC: Total cholesterol.

The parameters associated with oxidative stress (TAC, TOS and OSI) were comparable between two groups (Table-1).

No correlations were found between age and OSI and lipid levels (p>0.05). A significant correlation was found between OSI and cholesterol (p<0.001; r=0.596), TG (p<0.001; r=0.476); and LDL levels (p=0.001; r=0.318). However, no correlation was found between OSI and serum HDL levels (p=0.564; r=0.058) (Table-2).

Discussion

Hyperlipidaemia is an important risk factor for atherosclerosis and it also plays an important role in cardiovascular disorders. The increased risk may be associated with elevated blood cholesterol concentration which is associated with enhanced LDL oxidation.[6]

Lipids, proteins and nucleic acids are exposed to oxidative stress and the resulting oxidative damage occurs in cell elements, such as cell membrane, when the stress levels exceed defense capacity.[7,8] LDL oxidation is the initiator factor of atherosclerosis.[9] However, there is a complexity in oxidative stress and lipid interaction that is oxidative stress induced by hyperlipidaemia or lipid peroxidation induced by increased oxidative stress.

Bhalodia et al reported that hyperlipidaemia enhanced renal damage induced by ischaemia/reperfusion (I/R) in hyperlipidaemic rats by way of increasing lipid peroxidation and decreased antioxidant enzyme activities

as well as increasing inflammatory response.[10] It is widely accepted that the lipid peroxidation products mentioned above could induce oxidative stress and be involved in the pathogenesis of a number of degenerative diseases.[11] Moriel et al found that the LDL from hypercholesterolaemic and hypertensive patients has a higher sensitivity to oxidation and that the plasma lipid-derived hydroxy/hydroperoxide content is increased in these subjects as compared with normolipidemic and normotensive participants.[6] Csont et al. reported cardiac oxidative stress induced by hypercholesterolaemia in mice.[12] These studies show that there is complex interaction and multisystemic responses in the relationship between oxidative stress and hyperlipdaemia.

By increasing the number of patients in the present study setups, we think it would be useful to conduct an analysis that evaluates the relationship between lipid metabolism and oxidative parameters, or to detect the relationship between the measurements of lipid levels and oxidative parameters after antioxidant therapy that is especially intended to answer the question: does oxidative stress trigger hyperlipidaemia, or vice versa?

Conclusion

Hyperlipidaemia with incidence rates increasing with age has an important role in the etiology of cardiovascular diseases, is also significantly affected by oxidative stress. Protection against oxidative stress with lifestyle changes can show a beneficial effect on lipid metabolism at young ages and can lower the risk of CAD in older ages. In addition, the level of oxidative stress people are exposed to, can be detected with OSI follow-ups at young ages. Thus, preventive measures should be taken at an early age.

References

1. Verit FF, Erel O, Sav M, Celik N, Cadirci D. Oxidative stress is associated with clinical severity of nausea and vomiting of pregnancy. Am J Perinat 2007; 24: 545-8.
2. Galic S, Oakhill JS, Steinberg GR. Adipose tissue as an endocrine organ. Mol Cell Endocrinol 2010; 316: 129-39.
3. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab 2004; 89: 2548-56.
4. Erel O. A novel automated direct measurement method for total antioxidant capacity using a new generation, more s ABTS radical cation. Clin Biochem 2004; 37: 277-85.
5. Erel O. A new automated colorimetric method for measuring total oxidant status. Clin Biochem 2005; 38: 1103-11.
6. Moriel P, Plavnik FL, Zanella MT, Bertolami MC, Abdalla DS. Lipid peroxidation and antioxidants in hyperlipidemia and hypertension. Biol Res 2000; 33: 105-12.
7. Raha S, Robinson BH. Mitochondria, oxygen free radicals, disease and ageing. Trends Biochem Sci 2000; 25: 502-7.
8. Jensen SJK. Oxidative stress and free radicals. J Molecular Structure Theochem 2003; 666: 387-92.
9. Doi H, Kugiyama K, Oka H, Sugiyama S, Ogata N, Koide SI, et al. Remnant lipoproteins induce proatherothrombogenic molecules in endothelial cells through a redox-sensitive mechanism. Circulation 2000; 102: 670-6.
10. Bhalodia YS, Sheth NR, Vaghasiya JD, Jivani NP. Hyperlipidemia Enhanced Oxidative Stress and Inflammatory Response Evoked by Renal Ischemia/Reperfusion Injury. Int J Pharmacol 2010; 6: 25-30.
11. Noguchi N. Role of Oxidative Stress in Adaptive Responses in Special Reference to Atherogenesis. J Clin Biochem Nutr 2008; 43: 131-8.
12. Csont T, Bereczki E, Bencsik P, Fodor G, Görbe A, Zvara A, et al. Hypercholesterolemia increases myocardial oxidative and nitrosative stress thereby leading to cardiac dysfunction in apoB-100 transgenic mice. Cardiovasc Res 2007; 76: 100-9.