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Assessment of oxidative stress and lipid profile in a sample of patients with non-Hodgkin's lymphoma

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Abstract

Objective: The aim of the current study was to observe the profile of MDA and plasma lipids (Triglyceride, Cholesterol, HDL-Cholesterol, LDL-Cholesterol and VLDL Cholesterol) in patients with non-Hodgkin lymphoma (NHL). **Materials and Methods:** The research included 100 subject who were divided into two groups; the first as controls (50 in number) and the second as non-Hodgkin lymphoma patients (also 50 in number).Fasting blood samples were taken from each subject controls and patients (During beginning chemotherapy after 21 days from consumption the Dose) at the Unit of endocrine and tumors at Al Ramadi hospital, Iraq, all samples were non-smokers. Further 5 ml Peripheral venous blood samples were taken into tubes after an overnight fasting for at least 10 hours) for assay Sera were kept at -20 °C until utilized. The comparisons taken in this study was disease factor, gender, age and BMI. **Results:** A significant increase in serum levels (MDA and TG) of patients with NHL was found in all the comparisons made in this study (disease factor, gender, age, BMI). With respect to T.C, the levels had an insignificant fluctuation. It was often found that there was an insignificant decrease in patient levels (disease factor, gender, younger age group), With regard to HDL C, in most of the comparisons (disease, gender, BMI), its decrease was significant and it was not signify ant in comparisons (age). For all comparisons, however, LDL-C saw a non-significant rise. As far as the level of VLDL-C was concerned, its increase was significant for (disease, gender, BMI) and was not significant for comparison (age). **Conclusion:** The serum lipid profile can be useful in the diagnosis of the disease because the plasma lipid profile of patients with non-Hodgkin lymphoma is modified, Lipid peroxidation of patients with cancer can be critical in determining the activity and nature of the disease. The increase in lipid peroxidation and the propensity of antioxidants to increase in cancer are most likely due to an adaptive answer.

Keywords: Malondialdehyde, Lipid peroxidation, Lipid profile andNon-Hodgkin's Lymphoma

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Introduction

A community of heterogeneous malignancies of lymphoid origin, non-Hodgkin lymphoma (NHL) is the neoplasm with the second highest rise in incidence over the course of several years (1). While the etiology in general and the explanation for the recent increase are still unclear, evidence consistently indicates that, as occurs in autoimmune and chronic inflammatory disorders, chronic activation of the immune system increases the risk of (2,3).

Non-Hodgkin's lymphomas can occur at any age and are often characterized by larger than average lymph nodes, fever, and weight loss (4). In both plants and animals, lipid

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peroxidation is a well established cellular injury mechanism. This method, which leads to the development and ultimately the loss of membrane function and integrity of lipid peroxides and their by-products, is commonly believed to be involved in the pathogenesis of many human diseases (5)

The end products derived from peroxidation of polyunsaturated fatty acids and associated esters are malondialdehyde (MDA) and 4-hydroxy-2,3-nonenal (HNE). A broad variety of end products, including malondialdehyde, contain impaired lipid hydro-peroxides (MDA). On the other hand, oxygen-free radical mediated lipid peroxidation can lead to malignancy (6-8). Aldehydes are relatively stable in comparison to free radicals and are thus capable of diffusing inside or out of the cell and attacking targets distant from the location of original events triggered by free radicals. These aldehyde molecules are considered the ultimate mediators of the toxic effects of biological materials caused by oxidative stress(5) .

For cells to expand and proliferate, cholesterol is necessary via its uptake or de novo synthesis; normal mammalian cells satisfy their need for cholesterol. Cholesterol is a cellnon-essential nutrient since it can be synthesized de novo from acetyl-coenzyme A, in addition to being taken from the setting (acetyl-CoA)-(acetyl CoA)-(acetyl-CoA)-(acetyl-CoA). It is known that some cancer cell lines rely on exogenous cholesterol for their growth (9).

The objective of this study was to determine the concentration and lipid profile of MDA, including: (TC., TG., HDL-C, LDL-C and VLDL-C) in 50 NHL patients and 50 healthy subjects matched by age.

Materials and methods

Sampling

Fasting blood samples were taken at the Endocrine and Tumors Unit of Al Ramadi Hospital, Iraq, from each subject (50) controls and (50) patients (during initiation of chemotherapy after 21 days after intake of the dose), and they were all non-smoker. A further 5 ml of peripheral venous blood samples were taken into tubes for at least 10 hours after fasting overnight) for assay. Sera should be isolated at 200 /g for 15 min for measuring parameters after centrifugation. Before Sera were used, it was kept at -20 °C.

Determination of Lipid Profile and MDA

Serum concentrations of Total Cholesterol (TC) andTG were measured using enzymatic methods(10,11) .The concentration of (HDL-C) wascalculated using the technique stated by(12). The (VLDL-C) concentration was calculated from the TG concentration, and then the (LDL-C) concentration was calculated from the TC, HDL-C

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and VLDL-C concentrations using the Friedwald and Levy process.(13) Concentration of MDAwas performed using Beuge and Aust method(14) .

Statistical analysis

Statistical analysisFor all the classes, the mean value (M) and standard deviation (SD) were measured .By using the t-student measure, the mean valuesfor the research and control groups were compared .TheMinitab-16software has been used to analyze various variables, such asindividuals with and without NHL, gender, age, and weight .In addition, this program was usedto identify the standard deviation and deviation. Furthermore, to analyze the variations and conductmore statistical studies, T-test and ANOVA were used .When P was less than 0.05 ,the difference was deemed statistically important(15).

Results

The findings indicate that there was a significant ($p<0.05$) increase MDA, T.G. and VLDL-C and non significant ($p<0.05$) increase in serum LDL-C when compared to control On the other hand there are a significant ($p<0.05$) decrease HDL-C and non significant ($p<0.05$) decrease in serum T.C when compared to control.

Table1: Effect of NHL on MDA and Lipid Profile levels in human serum.

Parameters	Control (Mean±SD)	NHL (Mean±SD)
MDA (µmol/l)	0.9600± 0.5242	1.7669± 0.6954 *
TC (mmol/l)	4.132±1.118	3.927±1.078
TG(mmol/l)	1.5136±0.6037	1.9975±0.8123 *
HDL-C (mmol/l)	1.1029±0.2746	0.7324±0.4057*
LDL-C (mmol/l)	2.7139±0.6444	2.9058±1.0071
VLDL-C (mmol/l)	0.2550±0.1121	0.3178±0.1211*

*: Significant change in comparison with control at ($p<0.05$).

From table 2 , the results shows a significant increase of MDA , TG and VLDL-C ($p<0.05$) and non-significant increase in serum LDL-C ($p<0.05$) when compared to control Conversely there are a significant decrease ($p<0.05$) in both male and female serum HDL-C and non-significant decrease ($p<0.05$) in serum TC when compared to control.

Table2: Effect of Gender on parameters.

Parameters	Male (Mean±SD)		Female (Mean±SD)	
	Control	NHL	Control	NHL
MDA (µmol/l)	1.000±0.520	1.828±0.781 *	0.9141±0.5447	1.6827±0.6055 *
TC (mmol/l)	4.59±1.11	4.20±1.22	4.227±0.958	4.138±1.188

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TG (mmol/l)	1.425±0.468	2.047±0.617 *	1.3060±0.7305	1.7130±0.5277 *
HDL-C (mmol/l)	1.059±0.298	0.631±0.341 *	1.1300±0.1994	0.6958±0.3660 *
LDL-C (mmol/l)	2.875±0.905	3.101±0.864	2.6783±0.2733	2.2692±1.1391
VLDL-C (mmol/l)	0.2633±0.0886	0.3477±0.0902 *	0.2566±0.1756	0.3536±0.1151 *

*: Significant change in comparison with control at ($p<0.05$).

Three age ranges are shown in Table 3, A significant increase ($p<0.05$) in MDA and T.G. for all groups, and non-significant ($p<0.05$) increase in serum VLDL-C and LDL-C for all groups when compared to control Conversely there are a significant ($p<0.05$) decreases in serum HDL-C in 2 , 3 age groups and non-significant ($p<0.05$) decrease in 1 age group for HDL-C , but serum T.C decrease in 1 group and increase in 2 and 3 groups when compared to control .

Table3: Effect of Age on parameters.

Parameters	**Age 20-35 (Mean±SD)**		**Age 36-50 (Mean±SD)**		**Age 51-65 (Mean±SD)**	
	Control	**NHL**	**Control**	**NHL**	**Control**	**NHL**
MDA (µmol/l)	0.766±0.341	1.595±0.370 *	1.241±0.218	1.685±0.497 *	0.740±0.243	1.633±0.447 *
TC (mmol/l)	4.32±1.13	4.23±1.07	4.314±0.368	4.376±0.892	4.467±0.790	5.17±1.06
TG (mmol/l)	1.197±0.359	1.629±0.530 *	1.801±0.629	2.343±0.662*	1.801±0.442	2.371±0.713 *
HDL-C (mmol/l)	1.047±0.262	0.894±0.381	1.169±0.347	0.818±0.427 *	1.078±0.191	0.799±0.308 *
LDL-C (mmol/l)	2.606±0.680	2.764±0.693	2.632±0.471	3.094±0.789	2.905±0.209	3.478±0.911
VLDL-C (mmol/l)	0.2349±0.0850	0.293±0.102	0.2661±0.0757	0.337±0.106	0.260±0.114	0.367±0.117

*: Significant change in comparison with control at ($p<0.05$).

There was an influence of BMI on the parameters per the tables and a significant increase ($p<0.05$) in the normal weight group of MDA, both TG. In contrast to control, both LDL-C and VLDL-C groups, but not significant increase ($p<0.05$) in both LDL-C groups and MDA over weight group.On the other hand, there was a significant ($p<0.05$) decrease in HDL-C relative to control in than the normal weight group, but not a significant ($p<0.05$) decrease in the HDL-C overweight group and all T.C groups.

Table4: Effect of BMI on parameters.

Parameters	**Normal weight (Mean±SD)**		**Overweight (Mean±SD)**	
	Control	**NHL**	**Control**	**NHL**

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MDA (µmol/l)	0.616±0.234	1.628±0.499 *	1.208±0.432	1.553±0.418
TC (mmol/l)	4.10±1.00	4.15±1.03	4.606± 0.649	4.72±1.00
TG (mmol/l)	1.235± 0.624	1.877±0.611 *	1.483± 0.422	2.102± 0.561 *
HDL-C (mmol/l)	1.115±0.168	0.858±0.444 *	1.036±0.366	0.765±0.398
LDL-C (mmol/l)	2.413±0.387	2.765±0.788	3.056±0.542	3.471±0.829
VLDL-C (mmol/l)	0.2295±0.0841	0.325±0.103 *	0.2760±0.0762	0.3617±0.0873 *

*: Significant change in comparison with control at ($p<0.05$).

Discussion

Lipids are major components of the cell membrane that are important for different biological processes, including cell growth and benign and malignant tissue division. The carcinogenesis process may be attributed to low plasma lipid levels in the proliferating tissues and in blood compartments(16).Several studies have shown a strong correlation between low levels of cholesterol and cancer (17,18) Cholesterol metabolism shifts, including increased cholesterol synthesis and cholesterol ester accumulation in tumor tissues correlated with a decreasein serum high density lipoprotein cholesterol, have previously been reported in numerous neoplastic cell proliferation models, including hematological malignancies (19-23) .

In conformity with the results of Musolino et al. (24), 48 patients with newly diagnosed malignant tumors reported a decrease in total and HDL-C levels and also suggested that the lipid profile may be considered a useful biochemical and diagnostic marker in hematological cancers. In 66 individuals with hematological malignancies, Dessi et al. (21) reported statistically significant improvements in the lipid profile with respect to the HDL-C level, while the TC level remained constant.The findings were also consistent with the results of Guven et al (25) and Zhu et al (26), who observed significantly greater plasma and erythrocyte MDA concentrations in patients with malignant lymphoma comparison to the control group, and suggested that the antioxidant mechanism was damaged in malignant lymphoma.Oxidative stress is a major factor in the growth ofcancer and a crucial factor in the development of diseases (27).

In various forms of cancer, previous studies reported increased oxidative stress (28). There is sufficient evidence that shows that free radicals, oxidative damage and lipid peroxidation perform a role in the pathogenesis and development of different cancers.(29-31)

The most general way of measuring oxidative stress is to calculate the substances produced by oxidant reactions with biomolecules as a biomarker such as MD.(30 ,32) Oxidative stress biomarkers are clinically relevant and the examination of body tissue and blood is not sufficient to classify cancers such as breast cancer ,31) . (29

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Several studies have suggested that lipid peroxidation may be implicated in the promotion of tumors because reactive and toxic metabolites can be produced by this method .MDA can react with proteins, DNA, and many other biomolecules and thus change their function and structure as one of the most prevalent and essential aldehydes in lipid peroxidation. MDA evaluation of tissues and plasma has been commonly used in various cancers in recent years, besides other tumor markers, such as albumin, which we reviewed in a previous study as a common tumor marker (33).

The levels of total cholesterol tend to be impaired by malignancy .Reduced synthesis or enhanced catabolism is involved in the likelihood of decreasing cholesterol levels .Regarding the nature and correlation of serum cholesterol to cancer, though , requires long-term systematic measurements for cancer diagnosis at multiple points in time. An enhanced import of cholesterol into cancer tissue, which has also been reported in acute myeloid leukemia cells and in different solid tumors, may be one of the possible reasons, since rapidly propagating tumor cells presumably need cholesterol for new membrane synthesis. (34)Hypocholesterolemia may also occur due to elevated activation of the LDL receptor in cancer cells,(35 ,36) which usually regulates the breakdown of low-density lipoproteins, the main protein transporting cholesterol in human plasma cells(36).

In a number of experiments, the relation between HDL-Cholesterol and cancers has also been a topic .The epidemiological research by Kritchevsky et al. (37) indicated that the first symptom of a malignant disease in the early stages could be low HDL-Cholesterol. Lim et al. recently stated that high HDL-Cholesterol serum was associated with a reduced risk of all subtypes of NHL(38). These results include HDL-Cholesterol as a preclinical NHL predictor and based on its etiological contribution, provide a strong foundation for some further observational studies .Low serum HDL-Cholesterol has also been identified as an indirect indicator ofan increased risk of postmenopausal breast cancer in obese and overweight women(39).

Our results provide the first potential evidence that high HDL-C is due to the low risk of dose-responsive NHL growth .In the first chronic inflammation, the correlation was greatest and is known to decrease both serum HDL-C levels and its anti-inflammatory effects. (40 ,41)Therefore، low HDL-C could be an indicator for the severity of systemic inflammation and NHL risk caused by inflammation .On the other hand, high HDL-C can be NHL defensive on its own .Inflammatory responses irrespective of non-HDL cholesterol levels appear to be modulated by HDL-C(42) By inhibiting cytokine-induced production of endothelial cell adhesion molecules and by preventing the chemotactic behaveof monocytes and lymphocytes(43 ,44), HDL-C appears to modulate inflammatory responses independent of non-HDL cholesterol levels(42).HDL-C can also preserve the safety of lymphocytes from oxidative stress (45 ,46).

Conclusion

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In conclusion, during high oxidative stress, patients with non-lymphoma Hodgkin's were under considerable oxidative stress .Cytotoxic regimen, as demonstrated by an increase in MDA and some distortion of the parameters of the lipid profile relative to those of the controls .A physiological reaction to early undiagnosed cancer stages can indicate the inverse correlation between cancer and serum cholesterol.

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