Lipid Peroxidation, Prooxidant and Related Antioxidant Proteins in Various Types of Hyperlipoproteinemic Males and Control

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Abstract

Thirty nine hyperlipoproteinemic (HPLic) male subject aged (48-63) year not on any of the lipid lowering drugs, attending out patient clinic at Baghdad Teaching Hospital, were included in the present study, in addition to twenty two normolipidimic male subjects of matched age were included as control throughout this study.

The first part of this study was devoted to the classification of the HPLic subjects according to the serum lipid and lipoprotein profile following defined criteria.

The lipid parameter including total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) were investigated in serum of HPLic subjects included in the study. The classification was performed according to Frederickson's type, as twenty one hypercholesterlemic (type IIa), and eighteen hypertriglycerdimia (typeIV) HPLic subjects.

In the second part of the study, lipid peroxidation marker measured as malondialdehyde (MDA), free iron, ferritin, transrerrin (Tf) and ceruloplasmin (Cp) levels were measured in the serum of all subjects included in the study.

The results indicated that significant increase was observed in the serum levels of Tc, Tg and LDL in type IIa and Type IV HPLic compared to control, and a significant decrease in HDL level in serum of both HPLic group compared to control, while no significant difference in serum HDL levels between type IIa and type IV HPLic was found.

The mean MDA, free iron and ferritin levels in serum were found to be significantly higher in both types IIa and IV HPLic compared to control, also ferritin level in serum of type IV showed a significant increase over that of type IIa (257.3±22.7 vs. 223±19.2 mg/ml; $p<0.05$). On the other hand, no significant difference in mean MDA and free iron levels between both types of HPLic groups were found.

The mean Tf and Cp levels in serum of type IIa HPLic group showed a slight increase, yet not significant when compared to control, but a non significant decrease in Tf and Cp levels in serum of type IV HPLic group compared to control was found, however neither Tf nor Cp levels showed any significant difference between both HPLic groups. These results suggest the presence of oxidative stress injury in subjects with either type IIa or IV HPL, which could be due to high levels of LDL which is more susptible to oxidation and high levels of serum free iron which act as a prooxidant agent in its free form.

Introduction

A series of cytotoxic oxygen species plays an important role in various disease as associated with dyslipidemia causing cell damage including lipid peroxidation, inactivation of enzymes, alteration of intracellular oxidation-reduction state and damage to DNA[1, 2]

Lipoproteins are essential for the transportation of lipids in the vascular system. Inherited defects in lipoprotein metabolism lead to the primary condition of either hypo or hyper lipoproteinemia (HLP), a few individuals in the population exhibit inherited defects in their lipoproteins leading to the primary condition. In addition, diseases such as diabetes mellitus, hypothyrodism and coronary heart diseases are associated with secondary abnormal

lipoprotein patterns that are very similar to one or another of the primary conditions. Virtually ,all the primary conditions are due to defect at a stage in lipoprotein formation, transport or destruction, however not all of the abnormalities are harmful[3, 4].

Iron play an important role in many biological processes because it is an ideal O2 carrier and it is an essential cofactor for numorous iron containing compounds, in form of heme or noheme structure to limit the availability of free iron which contribute to free radical production via catalyzing Haber-Weiss and Fenton reaction[5]. Iron is taken-up by intestinal mucosa as Fe++ and converted to the trivalent form which bound to transferrin (Tf), aglycoprotein with two iron-binding sites, which transports iron to the liver or spleen where the fraction of unused and highly toxic iron is stored as ferritin molecules in order to be neutralized. A poferrintin, the protein fraction of ferritin, is spatially folded to create a central groove that holds oxidized iron molecules, the heavy chain in apoferritin molecule exerts ferroxidase activity, catalyzing the oxidation of ferrous to ferric ions, which prevents iron-induced cyclic redox reactions that would spread and amplify the oxidative damage to living cells. This activity occurs under aerobic conditions, allowing the storage of intracellular iron[6,7]. However ceruloplasmin (CP) (the copper carrying protein) seems to play a key role in the oxidation of ferrous iron and hence it releases from cells and loading onto apotransferrin, as the transferring binds largely the ferric form, so Cp is involved primarily in maintaining iron homeostasis and preventing iron-mediated free radicals injury, thus Cp is considered as antioxidant through its ferroxidase activity [8].

Hyperlipoproteinemia could increase levels of oxygen free radicals in various ways; hypercholesterolemia increases cholesterol content of platelets, polymorphonucleare leukocytes and endothelial and smooth muscle cells, neutrophils, nonocytes, and platelets may be the source of oxygen free radicals in hypercholesterolemia[9].

Antioxidant substance are used to keep free radicals under or at physiological control levels [10].

However, in the literature there were limited and conflicting data on the relationship between lipid peroxidation, iron as aprooxidant agents and some related proteins as antioxidant. Therefore this study was carried out to explore whether important proteins of iron metabolism are altered in subjects with various type of HPL, namely the ferritin, tansferrin, and cerulopasmin, since binding of iron to these proteins prevents, or greatly decreases participation of this metal in oxygen radical formation.

Experimental Part

Subjects

The present study was performed on males aged 48-67 years with different lipoprotein profile attending out patient clinic at Baghdad Teaching Hospital.

Noromolipidemic subjects were recurred from male subjects aged 45-65 years, who had plasma triglyceridws (TG)<203 mg/dl and total cholesterol (Ch)<217 mg/dl, according to criteria in Tietz (1999)[11]. No medication known to influence the lipid status was admistrated in any of

the subjects during the last six months. Patients with diabetes mellitus, hypothyrodism, nephritic syndrome or any other serious illness during the pervious six months were excluded. The classification of HPLic subjects was made according to Fredrickson's type, as following criteria in (Tietz 1999) [11] Type IIa HPL: plasma TG<203 mg/dl, total Ch>270 mg/dl and LDLc>131 mg/dl. Type IV HPL: plasma TG>203 mg/dl, total Ch<270 mg/dl and LDLc<131mg/dl.

Blood was drawn from subjects fasting (>12 hours) with type IIa (n=21), type IV (n=18) HLP and an age matched normolipidimic as control group (n=22).

In plain tubes 5ml blood was left to clot at room temperature for 15 minutes, centrifuged at 3500 rpm to separate serum from erythrocytes. Sera were used directly for some parameter measurements; the rest was divided in to small portions and kept frozen.

Lipid profile analysis

Fraction of serum lipids was performed by using ready kit from Bio Merieux A.S., France as follows:

Serum total Ch was determined by the Ch enzymatic method using a series of enzymes (i.e. Ch easterase, Ch oxidase and peroxidase). The chromagnic compelex formed was followed colorimetrically at soon, the absorbance of a known concentration of standard Ch was used to determine the concentrations of samples by proportion[12].

1- Serum TG was determined colorimetrically by TG enzymatic method using a series of enzymes (i.e. lipase, glycerokinase, glycerol-3-phosphate oxidase and peroxidase)[13].
2- Serum LDLc was estimated indirectly by the use of Friedewald FormulaSerum HDLc was determined after precipitation of chylomicron, VLDL and LDL contained in the serum samples by the addition of 4% phosphotungstic acid solution magnesium chloride, the supernatant obtained after centrifugation contains the HDL, from which the Ch was determined as described in [12].

Lipid peroxidation marker determination

Malondialdehyde (MDA), an end product of the breakdown of polyunsaturated fatty acids, reacts with thiobarbituric acid to give a red chromophor that has a maximum absorbance at 532 nm. In this method, protein was precipitated with trichloroacetic acid (70%) which also acidifies the medium; chloroform was added to remove the dispersed lipids then centrifuged. The absorbance of the supernatant was recorded at 532 nm against a blank. The MDA concentration was calculated by using molar absorbtivity coefficient of 1.56×105 L.mol-1.cm-1 [15].

Serum iron determination

The iron was determined in the serum by using a ready kit from TECO Diagnostics France. The iron is allowed to dissociate from its ferric transferrin complex by the addition of an acid buffer containing hydroxylamine. This process reduces the ferric iron to ferrous form. The chromogenic agent (i.e. Ferene) provided with the kit, will form a highly colored ferrous complex, which is measured spectrophotometrically at 560nm.

Transferring (Tf) determinatiom

The concentration of the Tf was determined indirectly as the ability of plasma protein to bind iron, the so called total iron-binding capacity (TIBC), where the unsaturated iron binding capacity (UIBC) is determined by the addition of a known ferrous salt concentration to the serum sample, so the added iron will bind to the unsaturated sites on transferrin. The excess (unbound) iron are reacted with ferene to form the colored complex and determined as in the method of serum iron determination using the ready kit TECO Dignostic France. The difference between the added iron and the amount of iron measured represents the unsaturated iron binding capacity; therefore the (TIBC) is determined by adding the serum iron value to the UIBC value. Then transferrin was estimated from the following equation[16]:

Transferring (mg/dl) = $0.7\times$TIBC (mg/dl).

Ferritin determination

Ferritin concentration in serum was determined by using a human ferritin test kit supplied by Bio Check Company U.K. The method based on solid phase enzyme linked immunosorbent assay (ELISA), where a system utilizes one rabbit anti-ferritin antibody for

the solid phase (microtiter wells) immobilization and a mouse monochlonal anti-ferritin antibody for the solid phase (microtiter wells) immobilization and a mouse monochlonal anti-ferritin antibody in the antibody-enzyme (horseradish peroxidase) conjugate solution[17].

Cerulopasmin (Cp) determination

The method for Cp concentration in serum based on the catalytic ability of Cp to oxidize the colorless P-phenylene diamine to a blue –violet oxidize form which has maximum absorbent at 525nm, using molar absorbtivity coefficient of 0.68 mol-1.cm-1 for the base[18] .

Statistical analysis

The results were expressed as mean ±SD of mean, using students t-test, significant variation is considered when P-values is≤0.05.

Results and Discussion

Lipids and lipoprotein parameters in all subjects participated in the study are shown in table (I).

Control (normolipidemic),type IIa HPL (hypercholesterolemic) and type IV HPL (hypertriglyceridemic) were classified according to the criteria mentioned in the experimental part (1).

All lipid parameters except for the high density lipoprotein (HDL) in both HPLic groups were significantly higher than of control group

Excess Ch is present in the form of low density lipoprotein (LDL) particles, so- called "bad Ch ", while the ratio of Ch in the form of (HDL), referred as "good Ch" to that in the form of LDL can be used to evaluate susceptibility for the development of heart disease , due to the binding of HDL to the esterified Ch released from the peripheral tissues and transfer cholestryl esters to the liver or tissues that use Ch to synthesize steroid hormones . The exact nature of the protective effect of HDL levels is not known; however, a possible mechanism is that a serum esterase which degrades oxidized lipids is found in association with HDL. Possibly ,the HDL-associated protein destroys the oxidized LDL (ox-LDL), accounting for HDL's ability to protect against coronary diseases . On the otherhand oxidized atherogenic lipoprotein, namely ox-LDL is taken up by immune-system cells (macrophages), which become engorged to foam cells. These foam cells would become trapped in the wall of the blood vessels and contribute to the formation of atherosclerosic plaques that cause arterial narrowing and lead to heart attacks [19].

The significant increase in serum LDL and the decrease in serum HDL in this study agree with results reported higher levels of LDL and lower levels of HDL in plasma of HPLic subjects ,they also claimed that LDL possess a direct toxic effect on endothelial cells , and the plasma concentration of ox-LDL might be one of the strongest predictors of endothelial dysfunction in early atherosclerotic lesions [20]. It has been

claimed that lower HDL values in plasma of HPLic subjects is to counter balance the ox-LDL levels ,because HDL not only attributed to

maintain normal cell cholesterol homeostasis by the reverse Ch transport, but also possess considerable antioxidant properties [21].

The results of lipid peroxidation marker MDA and free iron in serum of the three studied groups are shown in table (II).

A significant increase in MDA and free iron levels in the serum of both HPLic groups compared to control was found ,while no significant differences in the levels of both parameters between both HPLic groups was noticed .

The results of the present study agree with others studies reported higher plasma levels of MDA,ox-LDL and free iron in HPLic subjects , which was explained by the fact that the increased plasma concentration of lipids and lipoprotein (as substrate for oxidation) results in higher concentration of their oxidation products . It has been postulated that HPL could

increase levels of oxygen free radicals in various ways ; hypercholesterolemia increase Ch content of plateles, leukocytes and endothelial cells so that endothelial , smooth muscle cells , neutrophils and platelets may be the source of oxygen free radicals . It has been also demonstrated a higher oxidative strees in HPLic subjects who also showed clinical symptoms of endothelial dysfunction later indicating that ox-LDL plays the crucial role in early stages of atherosclerosis [20,22,23] .

Iron is intensively discussed as a possible risk factor of atherosclerosis, mainly for its catalytic role in free radical formation and subsequent oxidative modification of atherogenic lipoprotein namely LDL [20]. On the otherhand a non –significant increase in plasma free iron was reported in some subjects with HPL compared to control , yet an increased oxidative stress markers were reported , which was claimed that an alteration in the antioxidant defence system occur in response to various diseases and HPL [24] . Increased iron availability is , theoretically , expected to contribute to macrovascular disease because iron has an adverse effect on endothelium , and accelerates the development of atherosclerosis through the oxidation of atherogenic lipoprotein , LDL [25] .

Serum Tf, ferritin and Cp levels for type IIa and type IV HPLic groups with matched sex and age normolipidimic control are represented in table III.

A significant increase in serum ferritin levels in both HPLic groups compared to control group was noticed. Similar results were reported,
when analysing subjects with different types of HPL. The authors stated that it is still unclear whether elevated iron stores (ferritin) alone or plasma free iron alone or both play a crutial role in pathogenesis for atherosclerosis and cardiovascular risk developing in such subjects [20]. However , a possible explanation for the relation between serum free iron and ferritin comes from the fact that synthesis of apoferritin is induced at both the transcriptional and posttranscriptional levels by the presence of free iron , since iron can be released from ferritin by the action of many factors including reducing agent that convert ferric to ferrous forms , so the increase in the ferrous form downregulates the affinity of iron-regulatory element (IRE) binding protein (BP) for its IRE binding site in the s region of ferritin mRNA , leading to increase ferritin translation , also when the concentration of antioxidants are low ,the reducing potential and anaerobiosis progressively increase , facilitating a rapid release of iron from ferritin . Additionaly, the ferroxidase activity in the heavy chain of apoferritin is downregulated in this setting, decreasing the incorporation of iron into ferritin. The overall result of oxidative reactions is an increase in the availability of free iron from ferritin molecule as well as from other molecules (containing iron) undergoing degradation. These events, in turn, can enhance and amplify the process of generation of free radicals, causing cellular and tissue damage .The oxidative stress also downregulates the affinity of IRE for IRE-Bp. Thus, ferritin can act both as a source of iron , which induce oxidative stress , and as a mechanism that protects against the formation of highly toxic free radicals , which are capaple of inducing lipid peroxidation [26,27,28].

The levels of TF and Cp in serum of both HPLic subject groups were not significantly alterd from that of control group , thus the results of the present study agreed partly with a study reported a significant increase in plasma Cp levels and non significant differences in plasma Tf levels of HPLic subjects they also reported that the increase of Cp level was more pronounced in HPLic subjects with diabetes , due to the consideration of Cp is one of the positive acute –phase reactants , whose concentration increase upon different diseases and this increase come from an increase liver synthesis of Cp through the enhancement of ceruloplasmin mRNA transcription and/or a decrease in catabolism of this protein [29]. The results of the present study agree with reported data stated that neither Tf nor Cp in plasma of HPLic showed significant differences from that for normolipidemic subjects [22] .

Two important agents were reported to be responsible for extracelluar antioxidant activity , through their rotes in iron homostasis, these are the copper containing protein , Cp and the iron-binding protein Tf , as Cp convert reduced iron released from its storage site (ferritin)to the oxidized form by this way Cp allows iron to bind to its plasma transporter

protein (transferrin), while Tf carries iron to storage site , at this site , the iron is released from Tf and stored inside cells as ferritin, then Tf may be used again for further iron transport [30,31].

In conclusion, this study shows that both subjects with either type IIa or type IV HPL have enhanced lipid peroxidation in their serum. The elevated serum LDL concentration, which is more susptible to oxidation, in addition to high serum iron levels, may result in higher lipid peroxidation . However the decreased concentration of HDL and non-increased levels of Cp and Tf as antioxidant are not likely to be sufficient enough to counter higher reactive oxygen metabolites production in HPLic subjects, which may cause oxidative stress leading to cellular and molecular damages thereby resulting in development of cardiovascular diseases. Therefore further studies needed to clarify the relation between lipid peroxidation and other antioxidant parameters of the defense system to draw a conclusion as to whether the changes in this antioxidant are causes or results of increased oxidative stress.

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Table (1): Lipid and lipoprotein parameters in sera of type IIa, type IV HPLic groups and normolipidemic control

Parameters / Groups	TC mg/dl mean±SD	P	TG mg/dl mean±SD	P	HDL mg/dl mean±SD	P	LDL mg/dl mean±SD	P
Normolipidimic Control n=22	184.3± 11.2		123.7± 9.8		45.8±4.6		117.4±8.7	
HPL type IIa n=21	365.7± 14.8	P<0.05	179.2± 18.2	P<0.05	41.6±7.3	P<0.05	188.7±21.2	P<0.05
HPL type IV n=18	246.8± 12.6	P<0.05	378.9± 11.3	P<0.05	40.7±5.8	P<0.05	129.1±13.2	P<0.05
		*P<0.05		*P<0.05		*p<0.05		*p<0.05

* Represent P value between type IIa and IV HPL groups

Table(2) :Serum malondialdhyde and free iron in type IIa, type IV HPLic groups and normolipidemic control group

Parameters / Groups	MDA nmol/dl mean ±SD	P	Serum free iron µg/dl mean ±SD	P
Normolipidimic Control n=22	16.3± 3.9		98.8 ± 7.2	
HPLic type IIa n=2 1	28.9 ± 4.2	P<0.05	118.9 ± 9.3	P<0.05
HPLic type IV n=18	30.3 ± 6.5	P<0.05	120.2± 10.1	P<0.05
		*P>0.05		*P>0.05

* Represent P value between type IIa and IV HPL groups

Table (3) : Serum transferring , ferritin and ceruloplasmin in type IIa ,type IV HPLic groups and normolipidemic control group

Parameters / Groups	Transferrin µg/dl mean ±SD	P	Ferritin mg/ml mean± SD	P	Ceruloplas. mg/dl mean ± SD	P
Normolipidimic Control n=22	268.8 ±13.4		118.3±15.8		18.8±3.7	
HPLic Type IIa n=21	270.3±11.7	P>0.05	223.7±19.2	P<0.05	20.1±4.2	P>0.05
HPLic Type IV n=18	267.9±16.3	P>0.05	257.3±22.7	P<0.05	18.7±9.2	p>0.05
		*P>0.05		*P<0.05		*p>0.05

* Represent P value between type IIa and IV HPL groups

Table(4) HPLic groups and normolipidemic control group

Parameters / Groups	MDA nmol/dl mean ±SD	P	Serum free iron µg/dl mean ±SD	P
Normolipidimic Control n=22	16.3± 3.9		98.8 ± 7.2	
HPLic type IIa n=2 1	28.9 ± 4.2	P<0.05	118.9 ± 9.3	P<0.05
HPLic type IV n=18	30.3 ± 6.5	P<0.05	120.2± 10.1	P<0.05
		*P>0.05		*P>0.05

* Represent P value between type IIa and IV HPL groups

الاكسدة الفوقية للدهون؛ مسببات الاكسدة ومضادات الاكسدة البروتينية ذو العلاقة في انواع مختلفة من ارتفاع البروتينات الدهنية من الذكور

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الخلاصة

شملت الدراسة الحالية تسعة وثلاثين من الذكور يعانون من ارتفاع الدهون في الدم تتراوح اعمارهم بين (48–63) سنة لا يتعاطى أي منهم علاجا" خافضا" لدهون الدم والمراجعين في العيادة الخارجية لمستشفى بغداد التعليمي، فضلا عن إثنين وعشرين من الذكور ذي مستويات دهون طبيعية في أمصالهم مقاربين في العمر بوصفهم مجموعة سيطرة .

كرس الجزء الاول من الدراسة لتصنيف الذكور المصابين بارتفاع الدهون في الدم اعتمادا" على صورة الدهون والبروتينات الدهنية على موجب معيار محدد.

تم التحري عن صورة الدهون وشملت الكولسترول الكلي (Ch) ، والكليسريدات الثلاثية (TG)، والبروتينات الدهنية عالية الكثافة (HDL) ،والبروتينات الدهنية واطئة الكثافة(LDL) في أمصال الذكور المشاركين في الدراسة وقد صنفوا على أسس نوع Frederickson وظهر ان واحدا وعشرين مريضا يعانون من ارتفاع الكولستيرول نوع (IIa)، وثمانية عشراخرين يعانون من ارتفاع في الكلسيريدات الثلاثية نوع (IV).

تم في الجزء الثاني من الدراسة تعيين مستويات مؤشر فوق أكسدة الدهون عن طريق قياس المالون ثنائي الالديهايد (MDA)، والحديد الحر، والفرتيين ،والترانسفرين،والسيريلوبلازين في أمصال جميع الذكور المشاركين في الدراسة.

أظهرت نتائج الدراسة ارتفاعا" معنويا" في مستويات الكولسترول الكلي والكلسيريدات الثلاثية والدهون واطئة الكثافة، بينما وجد انخفاضا" معنويا" في مستوى البروتينات الدهنية عالية الكثافة في أمصال المجموعتين نوع(IIa) ونوع (IV) مقارنة مع مجموعة السيطرة،في حين لم تظهر فروقات معنوية في مستوى الدهون العالية الكثافة بين المجموعتين نوع(IIa) و (IV).

أظهرت معدلات المالون ثنائي الالديهايد والحديد الحر والفرتين ارتفاعاً معنوياً في المجموعتين نوع IIa ونوع IV مقارنة مع الاصحاء، كذلك وجد أرتفاعاً معنوياً في مستويات فرتين المصل في المجموعة نوع IV مقارنة مع المجموعة نوع IIa (22.7 ±257.3 مقابل 19.2±223 ملغم \مل علما إن $p<0.05$)، في حين لم تظهر فروقات معنوية في معدلات المالون ثنلئي الالديهايد ولا الحديد الحر بين المجموعتين IIa و IV.

أظهرت النتائج إرتفاعاً غير معنويً في مستويات الترانسفرين والسيروبلازمين في أمصال مجموعة النوع IIa مقارنة مع مجموعة السيطرة، كما وجد إنخفاضاً غير معنوي مستويات الترانسفرين والسيروبلازمين في أمصال مجموعة النوع IV مقارنة مع مجموعة السيطرة. بينما لم تظهر فروقات معنوية بالنسبة الى مستويات الترانسفرين والسيروبلازمين بين المجموعتين IIa و IV.

اقترحت هذه الدراسة وجود أضرار نتيجة للشد التأكسدي في الافراد الذين يعانون من إرتفاع دهون الدم سواء كانوا من المجموعة نوع IIa أو نوع IV الذي قد يعزى الى المستويات المرتفعة للدهون واطئة الكثافة التي تكون عرضة للاكسدة أكثر من غيرها من الدهون والمستويات المرتفعة للحديد الحر في المصل الذي يكون عاملا مولدا للاكسدة في صورته الحرة.