The Relationship between Uric Acid Concentration and Some of Plasma Lipids in Patients with C. V. Disease in General Hospital of Al-Nasseriya

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Abstract

Cardiovascular disorders are refer to the class of diseases that involve the heart or blood vessels (arteries and veins). While the term technically refers to any disease that affects the cardiovascular system. Cholesterol is classified as a sterol (a contraction of steroid and alcohol).Although cholesterol is essential for life; high levels in circulation are associated with atherosclerosis. Triglyceride (more properly known as triacylglycerol , TAG or triacylglyceride) is a glyceride in which the glycerol is esterified with three fatty acids. It is the main constituent of vegetable oil and animal fats.High-density lipoproteins (HDL) is one of the 5 major groups of lipoproteins (chylomicrons, VLDL, IDL, LDL, HDL) which enable lipids like cholesterol and triglycerides to be transported in the blood stream. In healthy individuals, about thirty percent of blood cholesterol is carried by HDL. Uric acid (or urate) is non-protein nitrogen compound with the formula C5H4N4O3. This study aimed to determine the relation between uric acid, cholesterol, triglyceride and HDL-cholesterol concentration in cardiovascular disorders patients. Serum sample were collected from 84 cardiovascular disorders patients attended to the general hospital of Alnasseriya. All samples were analyzed for uric acid , cholesterol, triglyceride and HDL-cholesterol concentration by enzymatic-colorimetric method kits. The data showed a highly significant increased in the serum concentration of uric acid , cholesterol and triglyceride in cardiovascular disorders patients compared with healthy control ($P<0.01$) .In addition , there was no significant increased in the serum concentration of HDL-cholesterol in cardiovascular disorders patients compared with healthy control ($P>0.05$) .The data of this study strengthen the possibility that high concentration of uric acid may act as a risk factor in cardiovascular disease.

Key words: high density lipoprotein (HDL-cholesterol),very low density lipoprotein(VLDL), low density lipoprotein(LDL),intermittent lipoprotein(IDL), hyperuricemia.

الخلاصة

تشير الإضطرابات القلبية الوعائية الى صنف امراض القلب او الاوعية الدموية (الشرايين والاوردة)بينما تقنيا تشير الى أى مرض يؤثر على الجهاز القلبي الوعائي . يصنف الكولوستيرول كأستيرول(إندماج الاستيرويد والكحول). بالرغم من أن الكوليستيرول ضروريُ مدى الحياة، ترتبط مستوياتة العالية في الدورة الدموية بمرض تصلب الشرايين.ترايكلسرايد(غالبا المعروف بتراي أسل كليسرول,TAG أو تراي أسل كليسرايد)هو كليسرايد الذي فية الكليسرول مؤستر بثلاثة احماض دهنية. وهو المكون الرئيسيُ للزيت النباتي والدهونِ الحيوانيةِ.في الافراد الاصحاء البروتينات الدهنية ذات الكثافة العالية (HDL)والتي هي واحدة من خمسة مجاميع من البروتينات الدهنية(chylomicrons, VLDL, IDL, LDL, HDL) التي تمكن الدهون مثل الكوليستيرول و الترايكليسرايد من الانتقال في مجرى الدم، حوالي ثلاثون بالمائة مِنْ كوليستيرولِ الدمِّ مَحْمُولُ مِن قِبل HDL.حامض اليوريك (أو اليورات) هو مُركّبُ عضويُ مِنْ المركبات النتروجينية غير البروتينية ذات الصيغةC5H4N4O3 . اجريت هذة الدراسة للتحري عن العلاقة بين تركيز حامضَ اليوريك وتركيز الكوليستيرول , ترايكلسرايد و HDL -كوليستيرول لدى مرضى الإضطرابات القلبية الوعائية حيث جمعت اربع وثمانون عينة مصل من مرضى الإضطرابات القلبية الوعائية الذين ادخلوا الى مستشفى الناصرية العام . جميع العينات فحصت بطريقة التحاليل اللونية –الإنزيمية, أظهرت النتائج فروق معنوية عالية في تراكيز حامض اليوريك, الكوليستيرول و الترايكليسرايد في مرضى الإضطرابات القلبية الوعائية مقارنة مع مجموعة السيطرة. بالاضافة الى ذلك لم تظهر النتائج فروق معنوية في تركيز HDL -كوليستيرول في مرضى الإضطرابات القلبية الوعائية مقارنة مع مجموعة السيطرة. نتائج هذة الدراسة دعمت احتمالية التركيز العالي لحامضِ اليوريك قَدْ يكون العامل الخطرِ في امراض الأوعية القلبيةِ.

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Received : 17 / 5 / 2009
Accepted : 27 / 10 /2009

Introduction

Cardiovascular disorders is any disorder that affects the heart's ability to function normally[1]. Cholesterol is a waxy material that is found in many food items including milk, cheese ,eggs, butter ,fish, beet, pork, chicken, goat meat, it is naturally occurring, fat-like substance with a complex chemical formula, and is used to build cells and make hormones.Itis present in plasma mainly esterified with fatty acids. The body can not break down the sterol nucleus, so cholesterol is either excreted unchanged in bile or converted to bile acids and then excreted. The effects of cholesterol on the heart may involve more than just one the arteries. There are some evidences unhealthy levels may affect the heart muscles and increase the risk for heart failure [2,3]. Triglycerides are composed of fatty acid molecules and are the basic chemicals contained in fats in both animals and plants[4]. High-density lipoproteins (HDL), the smallest and most dense. (Referred to as the "good" cholesterol) It is formed in the liver and walls of the small intestine, while maturing in blood stream, it obtains cholesterol from the surrounding tissues.The blood circulation then transports the HDL back to the liver , from where the cholesterol is excreted in the bile[5]. Uric acid is the major product of purine metabolism and is formed from xanthine by the action of xanthine oxidase. The normal limits of serum uric acid are between 387 and 416 µmol/L in men and lower than 327 µmol/L in women[6].Hyperuricemia is usually defined as a serum uric acid level of 416 µmol/L or higher in men, and 357 µmol/L or higher in women[7,8].

Methods

Studied group

This study included (84) patients from general hospital of Al-Nasseriya. Patients' ages ranged between (30-70) years .

Sample collection

From each patients included in this study blood samples were collected to obtain the serum during the period between May/2008 until November/2008. All cardiovascular disorders patients were diagnosed by consultants of cardiologist in hospital of Al-Nasseriya.

Procedure

Enzymatic -colorimetric method for detection of uric acid , cholesterol, triglyceride and HDL-cholesterol concentration in serum : uric acid , cholesterol, triglyceride and HDL-cholesterol: enzymatic –colorimetric method kits provided by (BIO LAB). Estimation of uric acid , cholesterol, triglyceride and HDL-cholesterol concentration in serum by enzymatic –colorimetric method **.**This method depend on that the chromogenic enzyme is binding with substrate. After incubation for 5min. at 37°C . The intensity of color developed is proportional to the concentration of the sample[9].

Statistical analysis

The suitable statistical methods were used in order to analyze and assess the results, includes the following [10]:

1- Descriptive statistics

A) Statistical tables including observed frequencies with their percentages.

B) Summary statistic of the readings distribution(mean,minimum&maximum).

2 – Inferential statistics

These were used to accept or reject the statistical hypotheses, they include the followings:

A) Chi-square (χ^2),

B) Repeated student test (t-test).

Note: The comparison of significant (P-value) in any test were:

S= Significant difference (P<0.05).

HS= Highly Significant difference (P<0.01).

NS= Non Significant difference (P>0.05).

3- Computer & programs

All the statistical analysis was done by using Pentium-4 computer through the SPSS program (version-10) and Excel application.

Results

The distribution of patients according to age groups is listed in table (1) shows that the age group between 51-60 years and 30-40 years is more percentage of cardiovascular disorders patients when compared with the healthy control give a non significant differences(p>0.05). Table (2) shows a non significant differences (P> 0.05) in the distribution of studied groups according to gender with predominance of the percentage of male patients than female patients . Data illustrated by table (3) clearly shows a highly significant increased (P<0.01) between the mean concentration of cholesterol and triglyceride in patients when compared with the mean concentration of cholesterol and triglyceride in healthy control, while no significant difference noticed (P>0.05) in between the mean concentration of HDL-cholesterol in patients and healthy control .In addition , the study shows a highly significant increased (P<0.01) in the mean of uric acid in cardiovascular disorders patients when the results compared with healthy control , as shown in table 4

Table 1: Distribution of studied groups according to age / Year .

			Studied groups		Total
			Healthy control	patients	
Age groups (year)	30-40	N	19	22	41
		%	38.0%	26.2%	30.6%
	41-50	N	17	20	37
		%	34.0%	23.8%	27.6%
	51-60	N	10	24	34
		%	20.0%	28.6%	25.4%
	61-70	N	4	18	22
		%	8.0%	21.4%	16.4%
Total		N	50	84	134
		%	100.0%	100.0%	100.0%

	Value	df	p-value
Chi_Square	6.958	3	0.073 NS

Table 2: Distribution of studied groups according to Gender.

			Studied groups		Total
			Healthy control	patients	
Gender	male	N	26	52	78
		%	52.0%	61.9%	58.2%
	Female	N	24	32	56
		%	48.0%	38.1%	41.8%
Total		N	50	84	134
		%	100.0%	100.0%	100.0%

	Value	df	p-value
Chi_Square	1.246	1	0.261 NS

Table 3 : Mean distribution of (Cholesterol, Triglyceride & HDL- Cholesterol) concentration in the studied groups .

		N	Mean	Std. Deviation	Minimum	Maximum	t-test (p-value)	Sig.
Serum Cholesterol (mmol/l)	Healthy control	50	3.964	.824	2.5	5.1	0.00	Hs
	Patient	84	5.383	1.185	1.3	7.0		
	Total	134						
Serum Triglycerides (mmol/l)	Healthy control	50	1.318	.349	.8	2.0	0.00	Hs
	Patient	84	2.252	.948	.9	5.5		
	Total	134						
S.HDL - Cholesterol (mmol/l)	Healthy control	50	1.273	.391	.9	2.0	.512	NS
	Patient	84	1.200	.433	.8	2.2		
	Total	134						

Table 4: Mean distribution of Uric acid concentration of studied groups.

Serum uric acid(µmol/L)	N	Mean	Std. Deviation	Range		t-test (p-value)	Sig.
				Minimum	Maximum		
Healthy control	5	291.33	108.17	137	416	.000	HS
Patient	8	375.14	88.59	120	520		
Total	13						

Discussion

Generally , this study showed the concentration of cholesterol and triglyceride was highly elevated in cardiovascular disorders patients compared with the healthy control groups. These findings are in a good agreement with other studies [11-15] who found that effects of increased cholesterol deposition in the arterial wall, enhanced foam-cell formation , generation of oxygen free radicals in monocytes, promotion of smooth-muscle–cell proliferation, and induction of monocyte chemotactic activity in endothelial cells .While hypertriglyceridemia may represent a procoagulant state mediated by increased levels of factors I,VII, VIII, and X and plasminogen activator inhibitor-1, as well as by reduced tissue plasminogen activator activity [16].Triglyceride-rich lipoproteins may also be directly atherogenic [17]. Researchers have suggested that the higher uric acid levels in subjects with cardiovascular disorders might be a compensatory response designed to counteract excessive oxidative stress[18,19] . This theory has a strong rationale in the biochemical characteristics of uric acid as anti-oxidant and is supported by pre-clinical studies performed *in vitro* and in experimental animals[20]. However, the role of uric acid in humans is still uncertain. The above results agreed with the results obtained by Skinner 1998 who observed that serum uric acid has antioxidant properties and contributes to free radical scavenging activity in human serum. When uric acid interacts with peroxynitrite to form a stable nitric oxide donor, vasodilatation increases and the potential for peroxynitrite-induced oxidative damage decreases [21]. Thus, uric acid can be protective against oxidative stresses, but it can also lead directly or indirectly to vascular injury[22,23].Others has been reported that uric acid promotes vascular smooth muscle proliferation and upregulates the expression of platelet-derived growth factor and monocyte chemoattractant protein-1 [24,25].Hypoxanthine is converted to uric acid via xanthine. This reaction can be catalyzed by xanthine hydrogenase and xanthine oxidase, the latter of which produces uric acid and superoxide. Thus, it is possible that, in certain diseased conditions, hyperuricemia is accompanied by the increased production of reactive oxygen species, which may result in the modulation of vascular contractility [26,27]. Another possible explanation is that hyperuricemia may induce endothelial dysfunction by decreasing the production of nitric oxide in the vascular endothelial cells [28] .Adenosine synthesized locally by vascular smooth muscle in cardiac tissue is rapidly degraded by the endothelium to uric acid , which undergoes rapid efflux to the vascular lumen due to low intracellular PH and negative membrane potential [29]. Uric acid synthesis is increased in vivo under ischemic conditions, and therefore elevated serum uric acid may act as a marker of underlying tissue ischemia. In human coronary circulation, hypoxia, caused by transient coronary artery occlusion, leads to an increase in the local circulating concentration of uric acid [30]. In conclusion, Raised serum uric acid concentrations are a powerful predictor of cardiovascular risk and poor outcome, although the underlying mechanisms remain unclear. Several potential explanations have been put forward to explain the apparent association between hyperuricaemia and cardiovascular risk. Studies have demonstrated mechanisms by which uric acid could be directly injurious to the endothelium and to cardiovascular function. Paradoxically, uric acid elevation could be expected to confer protective anti-oxidant effects in the cardiovascular system, but these potential benefits may be obscured by detrimental effects elsewhere. The effects of raising or lowering serum uric acid on endothelial function, autonomic regulation and progression of atherosclerosis require direct investigation, in order to understand a possible dual action in the cardiovascular system. Identifying the mechanisms by which uric acid interacts with cardiovascular regulation will give us greater understanding of the role of hyperuricaemia for individual patients and allow a more rational approach to treatments that modify serum uric acid concentration.

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