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Effect of Uric Acid and Lipid Profile on Myocardial Infarction in Iraqi Population

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Abstract

Myocardial infarction is a common presentation of coronary artery disease. This study was aimed to evaluate the effect of uric acid and lipid profile on myocardial infarction in Iraqi population.

The study was conducted on (30) patients acute myocardial infarction, and (30) apparently healthy subjects were taken as control group. The level of uric acid and lipid profile was determined using colorimetric method.

Patient group had significantly higher level of uric acid ($P < 0.01$), total cholesterol ($P < 0.01$), triglyceride ($P < 0.01$), low density lipoprotein ($P < 0.01$), very low density lipoprotein ($P < 0.01$) and significantly lower level of high density lipoprotein ($P < 0.01$) than control group. The results of this study also showed a significant difference ($P < 0.01$) between males and females regarding uric acid, total cholesterol, triglyceride, low density lipoprotein, very low density lipoprotein and high density lipoprotein in both study groups.

The body weight of the patients demonstrated as body mass index showed a significant association ($P < 0.01$) with the level of uric acid, total cholesterol, triglyceride, low density lipoprotein and high density lipoprotein in patient group. In conclusion, uric acid may have an effect on precipitating some risk factors of myocardial infarction.

Key words: Uric acid, lipid profile, myocardial infarction.

Introduction

Myocardial infarction (MI) means interruption of blood supply to a part of the heart, and it is almost always due to the formation of occlusive thrombus at the site of rupture or erosion of an atheromatous plague in coronary artery causing heart cell to die and without treatment the infarct related artery remains permanently occluded in 30% of patients (Bloomfield, *et al.* 2006).

Uric acid is a heterocyclic organic compound with the formula $C_5H_4N_4O_3$ and a molecular weight of 168 Daltons. Uric acid is the final metabolic product of purine metabolism in humans, and is excreted in urine (Ming, 2012). Uric acid produced from xanthine by the enzyme xanthine oxidase (Enomoto, *et al.* 2005).

Hyperuricemia has been shown to increase the risk of CHD related-events such as MI independently of other CHD risk factors, and is linked to higher mortality rates of CHD. A recent meta-analysis of a prospective cohort study showed that there is a 12% increase in mortality with each extra 1 mg/dL of uric acid in a person with CHD (Kim, *et al.* 2010).

The presence of hyperuricemia increases the risk of CHD by approximately 70% in women, but not in men (Wheeler, *et al.* 2005). Lipids are heterogeneous group of compounds which are water-insoluble but soluble in nonpolar solvents such as alcohol, ether, chloroform, benzene (Carl, A. and Edward R. 2008). Lipids are presented in the plasma as fatty acids, triglycerides, cholesterol and phospholipids, they can be transported as lipoprotein particles, i.e. associated with proteins (Smith, *et al.* 2000). lipids play crucial roles as coenzymes, electron carriers and emulsifying agents in the digestive tract, hormones and intracellular messengers (David L. and Michael M., 2005).

Lipoproteins are macromolecular complexes that carry various lipids and proteins in plasma (Ginsberg HN. 1998). Several major classes of lipoproteins have been defined by their physical and chemical characteristics, particularly by their flotation characteristics during ultracentrifugation (Young, *et al.* 2007). The lipids are mainly free and esterified cholesterol, triglycerides, and phospholipids. The hydrophobic triglyceride and cholesteryl esters compose the core of the lipoproteins, which is covered by a unilamellar surface that contains mainly the amphipathic (both hydrophobic and hydrophilic) phospholipids and smaller amounts of free cholesterol and proteins. Apo B100 is required for the secretion of hepatic-derived VLDL, IDL, and LDL. Apo B48 is a truncated form of Apo B100 that is required for secretion of chylomicrons from the small intestine (Merkel, *et al.* 2005).

Material and methods

The MI group who subjected to this study were (30) persons in the age group ranging from 38 – 90 years , the mean ± standard deviation (SD) was (60 ± 12.2 years). This group comprised of males (60%), with their age ranging from 38 - 70 years old, the mean ± SD was (55.5 ± 10.4 years) , and females (40%) with age ranging from 50 - 90 years, and mean ± SD was (66.7 ± 11.9 years).

Thirty apparently healthy individuals were taken as a control group of the age ranging from 38 – 79 years, the mean ± standard deviation (SD) was (57.87 ± 13.68 years). This group comprised of males (57%) their age ranging from 38 - 80 years , mean ± SD was (61.76 ± 13.57 years), and females (43%) their age ranging from 39 - 79 years , mean ± SD was (52.8 ± 12.5 years) .

The age and sex of this group were matched to age and sex of MI group, where statistical analysis showed non-significant differences in the age and sex between patient and control groups ($p > 0.05$).

A permission was taken from all subjects to contribute to this study after they were told about the aim and advantages of this study.

- The level of serum uric acid was determined by using colorimetric method mentioned earlier (Carl, A. and Edward R.2006).
- Cholesterol concentration was determined enzymatically according to the method described earlier (Allain, 1978).
- Triglycerides concentration was determined enzymatically according to the method described by Allain Fossati and Prencipe (Fossati P. and Prencip L. 1982).
- HDL level determined colorimetrically as mentioned earlier (Carl, A. and Edward R. 2006).
- VLDL concentration was calculated by dividing triglycerides value by 2.22 (Godkar, P. 1994).
- LDL concentration was calculated by using Friedewald equation (Godkar, P. 1994).

Determination of risk factors of the patient groups

Patients were considered hypertensive when they were already on antihypertensive treatments or their systolic blood pressure was ≥ 140 and diastolic blood pressure was ≥ 90 in two reading times with 24 hours interval (Kasper, *et al.* 2005). Patients were considered as diabetic when they had history of diabetes, on treatment including diet regime or when fasting blood glucose ≥ 7.0 mmol/L (126 mg/dl) or random blood glucose ≥ 11.1 (200 mg/dl) (WHO, 2006). Diabetic patients included in this study were of type 2 diabetes with disease duration of about 10-13 years.

Body mass index (BMI) is a ratio of a person weight to height; it commonly used to classify weight as healthy or unhealthy. BMI calculated as follow (Whitlock, *et al.*2005):

$$BMI\ (kg/m^2) = weight\ (kg) / height\ (m^2)$$

Results

Plasma concentration of uric acid was increased significantly in patients with MI when compared with control group ($P < 0.01$), as illustrated in figure (1).

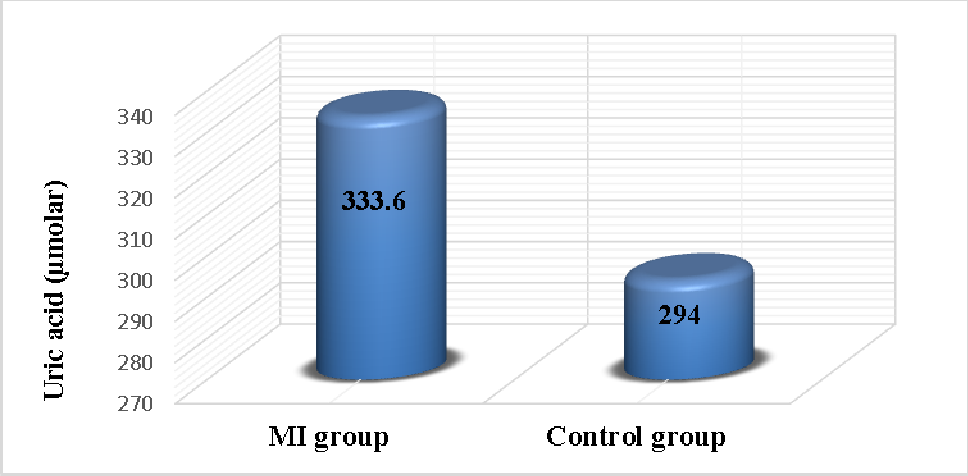

Figure (1) Plasma uric acid concentration (µmolar) in study groups.

The level of uric acid was compared for both genders, age and BMI groups for both study groups, as shown in table (1).

Table (1) the effect of gender, age and BMI on uric acid level in study groups.

Parameter	Subclass	Uric acid (µmolar) Mean ± SD	
		MI group	Control group
Gender	Male	374.9 ± 35*	303.3 ± 32.8
	Female	291.75 ± 25	281.8 ± 24.6
Age (years)	30-39 year	328 ± 0	292.9 ± 36.6
	40-49 year	334.1 ± 24.3	278 ± 33.5
	50-59 year	312.4 ± 39.7	279.9 ± 27
	60-69 year	320.7 ± 31.3	304.4 ± 17
	70-79 year	330.6 ± 31.7	317.5 ± 13
	80-90 year	325.1 ± 20.3	329 ± 0
BMI (Kg/m^2)	Under-weight	272.5 ± 24*	293 ± 0
	Normal weight	328.1 ± 36.8	289.3 ± 30.8
	Over-weight	395.2 ± 26.4	304 ± 34.5

*** Significant difference (P < 0.05). ** SD = standard deviation.**

The present study showed a significant difference in the level of uric acid between males and females in MI group ($P < 0.05$). There was no significant association between age and uric acid in MI group ($r = 0.2$, $P > 0.05$). The present study also showed that the over-weight individuals had significantly higher level of uric acid than normal or under-weight individuals in both study groups ($P < 0.05$).

To demonstrate the effect of risk factors of MI on uric acid concentration, the correlation between uric acid serum level of MI patients with each of these risk factors was tested, as shown in table (2).

Table (2) Effect of stroke risk factors on uric acid level

Risk factor / Parameter		Uric acid (µmolar)		O.R.	Confidence Interval at 95%	P
		Normal	High			
Hypertension	Normotensive	32 (52%)	30 (48%)	2.7*	1 – 7	S
	Hypertensive	8 (28%)	20 (72%)			
Smoking	Non-smokers	31 (46%)	37 (54%)	1.2	0.46 - 3.2	NS
	Smokers	9 (41%)	13 (59%)			
Diabetes mellitus	Non-diabetics	33 (48%)	30 (52%)	3.1*	1.2 - 8.5	S
	Diabetics	7 (26%)	20 (74%)			

* Significant difference ($P < 0.05$).

The table (2) showed a significant difference in the level of uric acid ($P < 0.05$) between hypertensive and normotensive patients in MI group. The table (2) also showed a significant difference in the level of uric acid ($P < 0.05$) between diabetic and non-diabetic patients in both stroke and MI groups.

The table (3) shows the mean level of serum lipids in study groups:

Table (3) Plasma concentration of lipid profile of study groups.

Parameter	Study group	
	MI group	Control group
Total cholesterol (mg/dl)	206.9 ± 39.7	160.6 ± 47.1
Triglycerides (mg/dl)	201 ± 84.6	141.3 ± 39.5
HDL (mg/dl)	34.4 ± 10.3	45.6 ± 8.8
LDL (mg/dl)	132.3 ± 41.5	86.7 ± 53.4
VLDL (mg/dl)	40.2 ± 17	28.3 ± 7.9
Non-HDL (mg/dl)	172.5 ± 40.4	115 ± 50.5

The table (3) showed that there was a significant difference between MI group and control group in all lipid profile parameters. The level of lipid profile was compared for both genders for each of the study groups, as shown in table (4).

Table (4) Effect of gender on lipid profile of study groups.

Study group / Parameter	MI group		Control group	
	Male	female	Male	female
Total cholesterol (mg/dl)	236 ± 14*	180.2 ± 16	179 ± 14.2	136 ± 16.2
TG (mg/dl)	217 ± 8.3*	192 ± 17.7	155.7 ± 16	122.6 ± 13
HDL (mg/dl)	22.7 ± 4.2*	46.9 ± 9.7	41.5 ± 6.5	51.2 ± 8.5
LDL (mg/dl)	167 ± 17*	95 ± 15.2	107 ± 16.1	60.4 ± 8.2
VLDL (mg/dl)	43.4 ± 5.6*	38.4 ± 4.2	31.1 ± 5.1	24.5 ± 4.8

*** Significant difference (P < 0.05).**

The present study showed that the levels of total cholesterol, triglyceride and LDL cholesterol were significantly lower in females than that of males, while the level of HDL cholesterol in females was significantly higher than that of males in both study groups (P < 0.05).
The association between BMI and lipid parameters was shown in table (6).

Table (6) Effect of BMI on lipid profile of study groups.

m Study group / Parameter	MI group		Control group	
	r	P	r	P
Total cholesterol (mg/dl)	0.42	S	0.53	HS
Triglycerides (mg/dl)	0.57	HS	0.61	HS
HDL (mg/dl)	-0.6	HS	-0.4	S
LDL (mg/dl)	0.41	S	0.39	S
VLDL (mg/dl)	0.3	NS	0.2	NS

r: Correlation coefficient. S: Significant difference (P < 0.05), HS: Highly significant difference (P < 0.01). NS: Non significant difference (P > 0.05).

The present study showed that total cholesterol, triglyceride and LDL had a significant positive association with BMI in both study groups, while a significant negative association was noticed between HDL and BMI in both study groups.

Discussion

Several mechanisms could cause the uric acid metabolic pathway to be a cardiovascular risk factor. Uric acid may stimulate vascular smooth cell proliferation, and reduce vascular nitric oxide production (Dawson, *et al.* 2007).

There are several suggested mediators of the deleterious effect of uric acid on cardiovascular health such as dysfunctional vascular endothelium; adhesiveness of platelets and granulocytes, subsequent release of cytokines and their effect on atherosclerotic plaques, and oxidative stress (Ambarish, *et al.* 2013).

A recent study found that hyperuricemia was significantly associated with poor outcomes in cardiac disease patients such as MI patients without chronic kidney disease, but not in hyperuricemic persons with renal failure (Filippatos, *et al.* 2011).

The effect of gender on uric acid level is well explained by the hormonal differences between males and females, since the estrogen have an impact on reducing the uric acid level in the blood (Woo, *et al.* 1994).

The association between BMI and uric acid may be explained by the fact that obesity affects urate metabolism, by decreasing urate clearance and increasing urate production (Omar, *et al.* 2007).

Uric acid has been suggested to play a role in the pathogenesis of early onset hypertension (Jules, 2014) but the levels may tend to dampen with age where stiffening of the aorta, activation of the renin-angiostensin system and renal vasoconstriction have a role to play (Teng, *et al.* 2011).

Chronic inflammation and endothelial dysfunction, as observed in patients with hyperuricemia, are likely to have participation in the mechanisms through which this substance may affect renal structure and operation leading to the genesis of hypertension (Zoccali, *et al.* 2006).

The relatively accepted explanation is that uric acid is a substance that its production increased by the organism to counteract the increased oxidative stress associated with diabetes mellitus thus, the increase of uric acid level in diabetes mellitus representing only a defense mechanism (Sérgio, *et al.* 2012).

Regarding lipid profile, Women have significantly different lipid and lipoprotein profiles than men regardless of age and menopausal status (Tremollieres, *et al.* 1999).

Presumably these differences are due to the different levels of circulating sex hormones, specifically estrogens and androgens in women versus men. It has been reported that women have higher production rates of Apo-I, the major HDL Apo protein, than do men, and that levels and production rates of Apo-I correlate well with estrogen level (Syed, *et al.* 2005).

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