

Nutrition Status and Serum Uric Acid Levels in Adult Men and Its Relation to Atherosclerosis

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Abstract

The study aimed to investigate the relationship between serum uric acid (SUA) concentration and atherosclerosis. Forty five adult men aged 40-60 years were volunteers from three different institutions in Cairo. They were divided into 3 equal groups: one group being clinically healthy and normal serum uric acid was taken as control. The second group suffered from hyperlipidemia and hyperuricemia. The third group suffered from hyperuricemia only. All groups were subjected to socioeconomic data collection, dietary and anthropometric (weights and heights) assessment, chemical analysis included serum levels of uric acid, lipid profile and platelets count. The results showed that all groups had their requirements from total calories and total proteins but low in carbohydrates. Group II had high fat and purine intake. It also showed positive correlation between serum uric acid and each of protein, fat and purine intake and the highest atherogenic index. Also there was positive correlation between serum uric acid and blood levels of triglycerides and cholesterol and negative correlation with blood platelets. Hence, it is recommended to have balanced diet with reasonable amount of foods rich in purine as prophylactic measure against atherosclerosis.

This was derived from MS thesis of Mrs Nawal M.El-Gohary from Faculty of Home Economic Helwan U 2005.

Introduction

It is well known that protein is an essential nutrient in human diets; however consuming excessive amounts can be hazardous to health. The general public may not be aware that high protein intake may be hazardous to health especially they are subjected to the massive mass media educational programs that lay stress on the importance of protein in the diet. The modern research is concerned with the medical problems caused by consuming too much protein where the excess more than actual need leaves toxic residues of metabolic waste in the tissues, causes auto toxemia over acidity accumulation of uric acid and purines in the tissues (**Michael, 2001**). Uric acid overproduction can also occur when there is excessive breakdown of cells which contain purines or

inability of the kidneys to excrete uric acid (*Donna, 2001*). Moreover excess proteins in the diet contribute to the development of many diseases such as arthritis, kidney damage osteoporosis, heart disease and atherosclerosis (*Michael, 2001*).

Aim of the study

The aim was to investigate the relationship between nutrition status and serum uric acid concentration and atherosclerosis in adult men; in particular to find the relationship between fat and protein intake and their effect on serum uric acid levels, lipid profile, and platelets count

Cases and methods:

Forty five adult men aged 40-60 years were chosen as volunteers from 3 different institutions: EL-Ahly sporting club, petroleum workers and outpatient's clinic from Om El-Masrieen general hospital in Cairo. Patients with renal impairment, diabetes or those who used to receive diuretics were excluded. After explaining the aim and procedure of the study they signed consent forms before volunteering

Group I: 15 clinically healthy individuals who had normal levels of serum uric acid were selected from Al-Ahly sporting club were considered as control group.

Group II: included 15 individuals working in petroleum companies and were complaining of hyperuricemia + hyperlipidemia

Group III: 15 individuals were selected from outpatients of Om El-Masrieen hospital were complaining of hyperuricemia only. All individuals were selected after performing, the biochemical analysis.

A predesigned questionnaire was used that included data on socioeconomic status (*Hussein et al., 1993*) diet history and food habits, 24 hours dietary recall and anthropometric, data (weights and heights). Body mass index (BMI) was calculated according to (*Garrow and Webstar, 1985*)

Those who had BMI 25-30 kg/m² and > 30kg/m² were considered over weight and obese respectively. Blood samples were collected after 12 hours fasting, then centrifuged at 4000 rpm for 20 minutes, serum was kept under -20°C till analysis. The following biochemical analysis were performed: serum uric acids according to *Fossati et al. ,(1980)* serum urea according to *Burtis and Ashwood (1999)*. Triglycerides according to *Titez (1982)*, total cholesterol (*Allain et al., 1974*). HDL-c *Lopez et al., (1977)* and LDL and VLDL (*Freidwal et al., 1972*).

Atherogenic index was calculated using the following equation in mg/dl

$$\frac{\text{Total CH-HDL-c}}{\text{HDL-c}}$$

Platelet count was determined as described by *Biggs (1972)*

Results and discussion:

The results revealed that all individuals were on jobs except in group III where 60% of them were jobless. Individuals of group I and III were distributed among the three levels of socioeconomic status where 26.7% and 40% were from low 60% and 40% from the middle and 13.3% and 20% were from the high class respectively, while all individuals of group II belong to the highest socioeconomic class. All of them were married except two from group II. The majority had below college education 73.3% and 60% for the men and 80% and 60% for their wives in group I & III respectively. While all individuals of group II and their wives had graduate education.

Dietary facts:

The majority of individuals in group I and III (86.7% & 80.0% respect) used to consume 3 meals a day, while in group II 46.7% used to consume 4 meals a day while the rest used to consume 5 meals daily. This group had the habit of eating meat or fish at lunch, most of the week and fried chicken at supper daily. *Joseph (2000) and Kuzuya (2002)* indicated that increasing number of meals a day is associated with an increase of serum uric acid. All group used to drink black tea in moderate amounts. Although tea contains low purines, *Saeng et al., (1995) and Bong et al., (1996)* claimed that tea inhibits Xanthine oxidase hence the conversion of xanthine to uric acid is limited. However, *Tang and Xia (1998)* found that serum uric acid levels are higher among those who used to drink tea than others who never had this habit. In our data the relation with tea was not manifest.

Our data also showed that all individuals in group II (hyperuricemia + hyperlipidemia) consume high fatty meat and poultry with skin compared with less than half amount in the other two groups. The majority of individuals in group II (93% used to consume spicy hot food which is almost double each of the other two group. *Bondy & Rosenberg (1980)* attributed the symptoms of gout among those who consume pickles to its high sodium chloride content that combines with uric acid to form monosodium urate which is responsible for the tophic deposits among the hyperuricemic cases.

Table 1 shows the consumption of energy and macronutrients calculated from the 24 hours recall for each of the three group. It is apparent that the first group (control) consume the least amount of energy compared with the other two groups. The consumption of fat and protein in group II shows significant high levels of fat $111.3 \pm 7.4\text{gm}$ and high level of protein $125.9 \pm 8.4\text{gm}$ which represent 119% and 120% of the requirements respectively followed by group III (hyperuricemic group). The least intake was reported by the control group. *Hugo and Joosens (1993)* reported positive correlation between serum uric acid and total protein intake. Group II also shows the least consumption of carbohydrates. This denotes that high fat together with high protein intake may be accused for the rise in serum uric acid and triglycerides in group II as well as total cholesterol, LDL-c, VLDL-c and the atherogenic index with low value of HDL-c as compared with the other two group (Table 2).

Table (3) shows the mean total amounts of purine intake by the three group as derived from their animal and plant sources. It is clear from the table that group II had the highest mean consumption of purine (815.5mg/day) which is highly significant as compared with the other two groups ($P < 0.001$). However, the table also revealed that the amount consumed by group II from food of highest purine and food of high purine was significantly higher than the other two groups ($P < 0.001$). Generally, foods that are considered as protein sources are the ones that have variable amounts of purine. Those who are considered as highest purine sources include sweetbreads which is the highest source (825mg/100mg) anchovies, sardines followed by livers, kidneys, brains, herrings and scallops which is the least (150mg/100gm) (*Bows and Church's 1970; Pennington 1989 and Rob et al., 1999*).

Brule et al., (1992) reported rise in serum uric acid on liver consumption. Also they reported rise in serum uric acid in eight normal persons fed haddock fish which is considered as higher purine source. *Emmerson (1991)* reported four reasons that contribute for high uric acid; excess purine consumption as a source of exogenous over production of urates, genetic mutation of purine reutilization enzymes, renal undersecretion of urates with normal renal function and the fourth factor is renal disease. In our cases we may attribute the rise in serum uric acid primarily to the first factor and possibly the second and third factors may have a role; but those who had renal disease were excluded from joining the study.

Table (4) shows the platelets count in the three groups. The table shows a drastic decrease in the platelets count in groups 2 and 3 to less than 50% compared with the control group ($P < 0.001$). According to *Christoph et al., (2002)* serum uric acid was first reported as a risk factor for atherosclerosis. Many epidemiologic studies suggested that there is an association between serum uric acid and cardiovascular disease. The authors postulated that uric acid may increase platelets adhesiveness and urate crystals that may lead to increased platelets lyses. Uric acid may also play a role in the formation of free radicals and oxidative stress. Thus, increased oxidative stress

appears to have an important role in development and progression of atherosclerosis (*Christoph et al., 2002*).

As a reflection of the higher energy consumption shown in table 1, we found higher body mass index for both group 2 and 3 (26.3 ± 2.0 , 27.7 ± 0.3 respect) as compared with the control 23.9 ± 0.3 and the difference was significant ($P < 0.005$) (Table 5). Although BMI in group 3 is higher than group 2, both of them are considered overweight. Hence it is important to advice the public to avoid being overweight and to be moderate in consuming foods rich in purines so as to avoid possible role in developing atherosclerosis.

Table (1): Mean values \pm SEM of energy, corbohydrate, fat and protein daily intake and % of RDA for the three groups .

Groups	Energy		Carb.		Fat		Protein	
	RDA		RDA		RDA		RDA	
	K. cal	%	gm	%	gm	%	gm	%
I	2632 \pm 163	94	348 \pm 85	91	89 \pm 6	96	109 \pm 7	104
II	2317 \pm 182	98	303 \pm 20	79	111 \pm 7	119	126 \pm 8	120
III	2754 \pm 180	99	372 \pm 22	97	90 \pm 6	97	113 \pm 8	108

Recommended dietary allowances, RDA (1989)

Table (2): Mean values \pm SEM of serum uric acid and lipid profile for the three groups .

Groups	Uric acid mg/dl	T.G mg/dl	Cholesterol mg/dl	HDL-C mg/dl	LDL-C mg/dl	VLDL-C mg/dl	AI*
Normal adults (group I)	5.4 \pm 0.4	109.5 \pm 4.8c	162.1 \pm 4.2	28.1 \pm 2.9	110.3 \pm 0.6	21.9 \pm 1.3	4.8 \pm 0.7
Hyperuricemia + Hyperlipidemia (group II)	8.4 \pm 0.67	425.3 \pm 12.8	403.6 \pm 10.2	17.6 \pm 1.9	302.2 \pm 8.9	83.7 \pm 2.7	21.9 \pm 8.9
Hyperuricemia (group III)	8.3 \pm 0.6	141.4 \pm 4.8	193.6 \pm 42	35.0 \pm 2.6	126.1 \pm 0.9	28.3 \pm 1.3	4.5 \pm 0.7

Total CH-HDL-C

*Atherogenic index = $\frac{\text{Total CH-HDL-C}}{\text{HDL-C}}$ (Steinberg and Witztum 1990)

Table (3): Mean \pm SEM of purine consumption according to their source for the three groups (mg/day)

Group	Food of highest purine	Food of high purine	Foods of low purine	Total
I M \pm SEM	25.5 \pm 4.7	298.3 \pm 11.7	69.3 \pm 0.8	393.1
II M \pm SEM	301.8 \pm 12.1	435.7 \pm 18.7	78.0 \pm 1.5	815.5
III M \pm SEM	12.7 \pm 4.7	210.1 \pm 9.7	97.7 \pm 2.5	320.5

Table (4): Mean values \pm SEM of serum uric acid and platelets count for the three tested groups

Groups	Uric acid mg/dl	Platelets count
I - Normal adults	5.4 \pm 0.4	280933 \pm 1902
II- Hyperuricemia + Hyperlipedemia	8.4 \pm 0.6	129000 \pm 870
III- Hyperuricemia	8.3 \pm 0.6	129800 \pm 874

Table (5): mean values \pm SEM of heights, weights and body mass index (BMI) for the tested groups

Group	Height (cm)	Weight (kg)	BMI*
Group I	171.9 \pm 12.0	70.7 \pm 4.3	23.9 \pm 0.3
Group 2	173.0 \pm 11.0	78.5 \pm 5.3	26.2 \pm 1.8
Group 3	170.3 \pm 11.5	80.2 \pm 5.4	27.7 \pm 0.3

Body weight (Kg)

$$* \text{ BMI} = \frac{\text{Body weight (Kg)}}{\text{Height in meters}^2} \text{ (Kg/m}^2\text{)}$$

Garrow and Webster (1985)

References

Allain, CC, Poon, LS. and Chan, CS (1974):

The enzymatic determination of total cholesterol. *J. Clin. Chem*, 20, 4.

Beiggs R. (1972):

Human blood coagulation haemostatic thrombosis, Blackwell Scientific Publication Oxford P590.

Bondy DK and Rosenberg LE. (1980):

Metabolic control and disease WB. Saunders Co. USA.

Bong J.A; Man JB and chung C. (1996):

Inhibitory effect of flavon 3-Ols isolated from oolong Science and Technology, 28 (6): 1084-1088B.

Bows and church's (1970):

Food Values of portions commonly used 13 ed. Philadelphia JB. Lipincott CO.,

Brule D; Sarwar G, Savoi I. (1992):

Changes in serum and urinary uric acid levels in normal Human subjects fed purine rich foods containing different amounts of adenine and hypoxanthine. *J. The American College of Nutrition*; 11 (3): 353-358.

Burtis, CA and Shwood ER (1999):

Tietz textbook of clinical chemistry 3rd ed Philadelphia WB Saunders Company P1383.

Christoph B; Hans JR, Stefan B. et al., (2002):

Serum uric acid as an independant predictor of mortality in patients with angiographically proven coronary artery disease. *Am. J. Cardiol*, 89: 12-17

Donna, R. (2001):

Uric acid; Univ. of Maryland Medical System Center, Baltimore p2.

Emmerson BT (1996):

The management of gout; *N England J. Med*, 334: 441-51.

Fossatti, P; Prencipe, Land Berti, G. (1980):

Use of 3,5 dichloro-2 hydroxybenzene sulfonic acid 14 amino Phenazone chromogenic system indirect enzymic assay of UA in serum and urine *Clin. Chem.*, 12/227-231.

Friedwald WT, Levy RT and Fredickson, DS (1972):

Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge, *Clinical Chemistry* 28: 499-502.

Garrow and Webster (1985):

Queslet's index (W/H²) *Eur J. Obes.* 9: 147-153.

Hussein, MA, Hassan, HA and Noor EF (1993):

National food consumption study final reports national nutrition institute, MOH, Cairo

Joseph M. (2000):

High uric acid linked to heart disease *Weekly Health Newsletter* Issue 153, may 14.

Kuzuya, K. (2002):

Effect of aging on serum uric acid level. *J. of Gerontology* 57A: M 660-M665.

Lopez-Virella, MF; Stone, P. Ellis, S. and Colwell J. A. (1977):

Cholesterol determination in high density lipoproteins reported by 3 different methods *J. Clin. Chem.*, 23: 992.

Michael, D. (2001):

Disease free living through fitness and nutrition <http://www.all-org/cb/a-protein.html>.

Pennington J. (1989):

Food value of proteins commonly used 15th ed. Philadelphia: JB Lippincott, Academy of Science by National Academy Press Washington.

RDA, Recommended Dietary Allowances (1989):

Food and Nutrition Board National Research Center Washington DC 10th ed.

Rob, L., Pat, R., June K. et al., (1999):

Purine yielding foods. *The Dalwation of America.*

Saeng GY, Yeung BP and Yeung HP, (1995):

Inhibition of xanthine oxidase by tea extracts from green tea. Oolong tea and Black tea. *U. Korean Soc. Food Nutrition* 24 (1): 154-159.

Steinberg D. and Witztun JL. (1990):

Lipoprotein and atherosclerosis: current concept *JAMA* 264: 3047-3052.

Tang J. and Xia O. (1998):

Influence of dietary habits and body weight on blood uric acid in the elderly human *Kuta Hush* 23 (5): 447-449.

Titez NW, (1982):

Fundamentals of clinical chemistry, Pub. WB Saunders Company P 492-503.

الحالة التغذوية وعلاقة مستوى حمض البوليك فى السيرم مع تصلب الشرايين لدى الذكور البالغين

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الملخص العربى

استهدفت الدراسة معرفة العلاقة بين مستوى حمض البوليك فى السيرم واحتمال حدوث تصلب للشرايين، تناولت الدراسة ثلاثة مجموعات كل مجموعة مكونة من ١٥ فرد. الأولى ضابطة والثانية تعاني من ارتفاع حمض البوليك ودهنيات الدم، والثالثة تعاني من ارتفاع حمض البوليك فقط وتم تجميع بيانات الحالة الاجتماعية والاقتصادية والاستهلاك الغذائى والمقاييس الجسمانية (أطوال وأوزان) والتحليل المعملية التى شملت الهيموجلوبين وعدد الصفائح الدموية – ومعدل تركيز حمض البوليك ودهنيات الدم وأسفرت النتائج على الآتى:-

* تناولت الثلاث مجموعات احتياجاتهم من السعرات والبروتين وقليل من المواد النشوية أما المجموعة الثانية خاصة فكان استهلاكها للدهون والبيورين كبير.

* يوجد ارتباط ايجابى بين حمض البوليك فى السيرم وكل من البروتين والدهون والبيورين المتناول وارتباط ايجابى بين حمض البوليك فى السيرم ومستوى الدهون فى الدم وارتباط عكسى مع الصفائح الدموية لذا ننصح بتناول غذاء متوازن وعدم الاسراف فى تناول المواد الغذائية الغنية بالبروتين.