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Evaluation of relationship between hyperuricemia and lipid profile

Dr. Sushil Kumar[1*], Dr. Ayan Banerjee[2], Dr. Mala Mahto[3], Dr. Alok Ranjan[4], Dr. Mamta Singh[5], Roshan Takhelmayum[6]

[1] Assistant Professor, Department of Biochemistry, All India Institute of Medical Sciences (AIIMS), Patna. Bihar, India

[2, 3] Associate Professor, AIIMS Patna, Bihar, India

[4] Associate professor, Department of Internal Medicine, AIIMS, Patna, Bihar, India

[5] Senior Resident, Department of Biochemistry, AIIMS, Patna, Bihar, India

[6] Tutor, Department of Biochemistry, AIIMS Patna, Bihar, India

Abstract
The relationship between serum uric acid and dyslipidemia is also complex and not fully elucidated. In recent years, atherosclerotic diseases and Coronary artery disease (CAD) have become important causes of mortality and morbidity in patients with gout. The objective of our study was to investigate the independent relation between serum uric acid and lipid profiles.

This study had been conducted in the Department of Biochemistry AIIMS Patna on adult patients who were referred from Out-Patient Departments (OPD). Relevant demographic, personal, medical and surgical details of the patients are recorded in the department during their enrolment for biochemical investigation. All the blood samples were analysed on fully automated clinical chemistry analyzer, Beckman Coulter AU 480.

Total 633 blood samples reports records were analyzed retrospectively from records for this study. Our study showed significant higher levels of uric acid in dyslipidaemic subjects. Since dyslipidaemia predicts the risk of CAD, it is important to consider uric acid levels in these patients for more comprehensive strategic management of risk factors and Visa-versa, while establishing the diagnosis of hyperuricemia, clinical suspicion of coexistent dyslipidaemia should also be considered.

Keywords: dyslipidaemia, hyperuricemia, lipid profile

Introduction

Hyperuricemia [elevated level of uric acid] is the consequence of increased uric acid production or and decreased excretion. It is also associated with glucose intolerance, hypertension and dyslipidemia, a cluster of metabolic and hemodynamic disorders which characterize the so-called metabolic syndrome [1]. Normal Uric acid levels are 2.6 to 6.0 mg/dl (0.155 to 0.357mmol/L (female) and 3.5 to 7.2 mg/dl (0.208 to 0.428mmol/L (male) [2].

Elevated SUA has been associated with increased cardiovascular morbidity and mortality in the general adult population, among subjects with a low cardiac risk (i.e, patients who were not obese or hypertensive, or had hyperlipidemia, diabetes mellitus, or metabolic syndrome) [3, 4]. It is unclear whether elevated uric acid levels are independent risk factors, or simply markers, reflecting the association between uric acid and other traditional risk factors such as blood lipids, metabolic syndrome, diabetes, and chronic kidney disease [5, 6, 7].

Hyperuricemia is said to be a mediator of proinflammatory endocrine imbalance in the adipose tissue which may be one of the factors for dyslipidemia and the inflammatory process leading to atherogensis. Animal model study have proved that uric acid causes dramatic increase in the expression and release of MCP-1, infiltration of macrophages and expression of proinflammatory cytokine TNF-α in the vascular smooth muscles, and thus can eventually lead to atherosclerosis [8].

Therefore, to assess the potential relationship between UA and lipid metabolism, we retrospectively analyzed results of a lipid profile include-total cholesterol (TC), Low-density lipoprotein cholesterol (LDL-C), High-density lipoprotein cholesterol (HDL-C), Triglycerides (TG), and Total cholesterol to HDL-C ratio (TC/HDL-C), in a large unselected adult outpatients.

Methodology

After obtaining ethical clearance from the institutional ethical committee for human studies of AIIMS Patna, this study had been conducted in the Department of Biochemistry AIIMS Patna on adult patients who were referred from Out-Patient Departments (OPD). Relevant demographic, personal, medical and surgical details of the patients are recorded in the department during their enrolment for biochemical investigations.

Total 633 blood samples reports of adult patients (21-80 years) were analyzed retrospectively from the records (available in the department) to retrieve results of UA, TG, TC, HDL-Cholesterol for this study. Neither inclusion nor exclusion criteria were applied to stratify the entire population of outpatients.

Fasting venous blood was collected, after centrifugation, all the blood samples were analysed on fully automated clinical chemistry analyzer, Beckman Coulter AU 480. The estimation of Uric acid, Triglycerides, Cholesterol, and HDL were done Uricase/peroxidase enzymatic method, GPO-PAP, Cholesterol oxidase, direct HDL-C two reagents homogenous system method respectively. LDL-C and very low density lipoprotein cholesterol (VLDL-C) were calculated by the traditional formula of Friedewald *et al*. [9].

We defined hyperuricemia by plasma uric acid concentrations greater than 7.0mg/dl (0.42mmol/L) in men or greater than 6.0mg/dl (0.36mmollL) in women [10].

Results & Discussion

Results of UA, and the other lipid variables were retrieved for 633 outpatients (21-80 years old) from records which is maintained in department of biochemistry. Out of 633 subjects, 8 male and 6 female patients had been removed from study because of high levels of triglycerides (>400 mg/dl) as Friedewald equation [9] can be applied to calculate LDL-C and VLDL-C.

Table 1: Age and Sex of Patients

Age	21-80 years
Male	337
female	282
Total	619

Table 2: Lipid Profile in a study Population of Outpatients Stratified According to Gender and Values of Uric Acid in Plasma

	Men			Women		
UA (mg/dl)	**<6.9mg/dl**	**>6.9mg/dl**	**P***	**<6mg/dl**	**>6mg/dl**	
No. of patients	296	41		238	44	
Age (years) ± SD	52.42±13.6	53.61±14.35	0.53	47.68±	51.20±	0.05
Triglycerides (mg/dl) ±SD	149.35±63.85	151.60 ± 50.60	0.82	139.81± 62.32	152.59 ±55.0	0.20
Total cholesterol (mg/dl ±SD	172.48 ±42.44	172.31±41.08	0.98	178.17±38.97	180.34±40.51	0.73
HDL (mg/dl) ±SD	43.73±9.32	39.83±6.53	0.01	45.45±9.85	44.41±9.71	0.52
Total cholesterol/HDL ratio ±SD	4.06±1.14	4.39±1.18	0.08	4.08±1.19	4.15±1.01	0.72
LDL (mg/dl) ±SD	98.87±35.82	103.46±36.47	0.58	104.75±34.19	105.40±36.56	0.90

P*-value for t-test for differences of mean for equal variance

Showing a positive correlation of uric acid and triglycerides, CHOL/HDL ratio, and LDL-C. Negative correlation observed between uric acid and HDL-C.

Table 3: Percentage of Hyperuricemic Subjects with Abnormal Values of Blood Lipids According to the Current NCEP [11].

	Men		Women	
UA mg/dl	**<6.9mg/dl**	**>6.9mg/dl**	**<6mg/dl**	**>6mg/dl**
No. of patients (633)	296	41	238	44
Triglycerides >150mg/dl	34% (100)	46% (19)	31% (75)	40% (18)
Total cholesterol >200mg/dl	23% (69)	34% (14)	26% (61)	73% (32)
HDL cholesterol <40mg/dl	37.5% (111)	49% (20)	28% (66)	32% (14)
Chol/HDL ratio >3.5	66% (197)	78% (32)	65% (155)	70% (31)
LDL >130mg/dl	19% (57)	21% (09)	19%; (46)	29% (13)

In Hyperuricemic subjects, almost half of the male patients were involved with hypertriglyceridemia and also have low HDL-C value; in about 70% of both gender had high Chol/HDL ratio.

Bendek *et al*. 1967 and other various studies have proved the association hypertriglyceridemia in hyperuricemia [12]. It has been shown in various other studies an inverse relationship between HDL and uric acid. The negative correlation of HDL in hyperuricemia resulted in decrease in good cholesterol (HDL) and consequently and increase in atherosclerosis and eventually predisposes to Cardio Vascular Disease. LDL-C is always considered to be bad cholesterol and increase in LDL results in atherosclerosis which leads to various cardiovascular events ranging from angina to myocardial infarction [13, 14, 15].

A study conducted (Madumita *et al*.) on 1485 Assamese subjects, an average healthy urban Assamese population has a TC, TG, HDL and LDL value of 170 mg/dl, 110 mg/dl, 40 mg/dl and 103 mg/dl respectively. They reported hyperuricemia were associated with dyslipidemia. The study documented a significant positive correlation ($p < 0.005$) between hyperuricemia and TC, TG and LDL and a significant negative correlation ($p < 0.005$) between hyperuricemia and HDL. Therefore, it is inferred that hyperuricemia is associated with dyslipidemia in Assamese population as is for other ethnic groups and races proved by various studies, and these hyperuricemic are at a high risk for developing CVD [16].

High levels of plasma triglycerides are related to hyperuricemia, the mechanism for this can be explained on the basis that triglycerides synthesis accelerates the de novo synthesis of ribose-5-phosphate to phosphoribosyl pyrophosphate (PRPP) through the common metabolic pathway of NADP/ NADPH, and as a result, uric acid production increases [17].

Hyperuricemic hyperlipidemic patients have a decreased renal excretion of urates compared with patients with hyperuricemia only [18].

Our results are consistent with the hypothesis that the association between UA and lipids variables. In men higher values of TG, CHOL/HDL ratio, & LDL-C, (statistical not significant), low value of HDL-C in hyperuricemic subjects (statistical significant); hyperuricemia in women was characterized by lower levels of HDL-C and higher values of triglycerides & CHOL/HDL-C ratio. Results of previous investigations highlight that an unclear relationship exists between UA and lipid metabolism besides hypertriglyceridemia, aq1most controversies arising from the lack of stratification for gender [19].

Conclusion

Detection of dyslipidemia at a preliminary level and its proper management will be able to prevent the morbidity and complications of CVD. Positive correlation between hyperuricemia and dyslipidemia, as illustrated in our study, may facilitate the claim that serum uric acid could be consider as a simple and economically viable biomarker in CAD patients.

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