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ORIGINAL ARTICLE

ROLE OF INCREASED SERUM URIC ACID IN STROKE

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ABSTRACT: The role of hyperuricemia as a risk factor for acute stroke is debatable as it is not clear whether Serum Uric Acid (SUA) promotes or protects against the cerebrovascular disease. In this study, the serum uric acid level and other risk factors like lipid profile, S. Adenosine Deaminase was estimated in patients with acute stroke and their relationship with ischemic stroke, and the association of factors like age, sex, obesity and smoking in the development of ischemic stroke. **METHODS:** A prospective, randomized study conducted on 100 cases of ischemic stroke presenting to Department of Medicine, GMC, Amritsar. Risk factors for stroke were noted such as hypertension, diabetes mellitus, metabolic syndrome, smoking, and obesity. Serum uric acid levels were measured in cases and controls. In addition the measurement of other risk factors likes lipid profile, Fasting blood sugar and S. Adenosine deaminase was done. The collected data were analyzed using Student's t-test. **RESULTS:** Out of 100 patients, 69 were males and 31 were females. The hyperuricemia and ischemic stroke was more in males than females (46 vs. 23). Hyperuricemia and ischemic stroke was associated with higher FBS (mg/dl) 122.33±18.46 and 129.37±15.40, HbA1c (mg/dl) 6.87±2.1 and 6.82±6.21, S. Triglyceride (mg/dl) 162.34±14.08 and 154.34±10.12, S. Total Cholesterol (mg/dl) 169±12.10 and 160.09±14.10, S.HDL-C (mg/dl) 42.11±10.17 and 45.16±11.40, S.LDL-C (mg/dl) 119.85±14.07 and 110.42±12.67, S.ADA (U/L) 45.62±7.90 and 49.47±9.57 at admission and at discharge respectively compared to the control. **CONCLUSIONS:** Hyperuricemia and ischemic stroke was significantly higher in patients with hypertension, Diabetes mellitus, metabolic syndrome, chronic smokers and BMI > 25 kg/m2. So control of these factors should be done to prevent future onset of ischemic stroke.

KEYWORDS: Hyperuricemia, Acute ischemic stroke; adenosine deaminase.

INTRODUCTION: Stroke is the third commonest cause of death in the world after coronary heart disease and cancer, especially in the elderly.[1,2] The mortality rate of stroke in the acute phase is as high as 20% and it remains higher for several years after the acute event in stroke patients than in the general population. Stroke, both ischemic and hemorrhagic, is a common and devastating disorder and currently one of the leading causes of mortality worldwide. It is an important cause of disability and dementia in adults aged ≥ 65 years and approximately 25% of stroke survivors develop dementia.[2] Several large studies have shown conflicting results regarding the clinical significance of elevated serum uric acid levels in cardiovascular or cerebrovascular diseases. Studies including the NHANES study concluded that uric acid is an independent risk factor for development of cardiovascular and cerebrovascular diseases.[3] The mortality rate of stroke in the acute phase is as high as 20% and it remains higher several years after the event of stroke.[2] Uric acid is the catabolic product of purine metabolism in humans. Uric acid levels are influenced by age and sex. Prior to puberty, the average serum uric acid is 3.6 mg/dl in males and females, however, after puberty the value reaches to adult levels with women having 1 mg/dl less than men. The lower level in women is due to an estrogen mediated increase of renal urate clearance.[4] However, it has been reported that

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increased levels of uric acid are associated with cardiovascular risk factor such as elevated serum triglyceride and cholesterol concentration, hypertension, obesity, and metabolic syndrome.[2,5] On the other hand various studies have established that uric acid acts as a neuroprotective agent due to free radical scavenging effects.[5,6]

The relationship between uric acid and stroke prognosis is ambiguous. Some studies have explored this relationship in acute stroke, but with different results. An experimental study in the rat brain showed that uric acid after thromboembolic stroke is neuroprotective.[7] A retrospective analysis of 2495 indoor patients with ischemic stroke in Glasgow scale suggested that high serum uric acid on admission predicted poor outcome.[8] Since serum uric acid (UA) is strongly associated with cardiovascular risk factors, it has been debated whether serum UA is a stroke risk factor or whether UA may be simply "marking" subjects with other, causal risk factors. Therefore, this study was planned to determine serum uric acid levels in patients with acute stroke and assess its relationship with cardiovascular risk factors.

MATERIAL AND METHODS: It was a population-based prospective and randomized study done in 100 patients diagnosed as ischemic stroke admitted in Department of Medicine, GMC Hospital, Amritsar, Punjab, India. The consent of the patients and ethical approval from the institutional ethical committee was taken. The patients were divided into two groups as Group I (control): Patients having S. Uric acid <7 mg/dl in men & <6 mg/dl in women and Group II (cases): Patients having S. Uric acid above these levels. Stroke was defined as per the WHO (World Health Organization) definition, "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin".[9] Only CT brain or MRI brain proved cases of acute ischemic stroke were included in he study. Normal Uric acid levels are 2.4-6.0 mg/dl (female) and 3.4-7.0 mg/dl (male). SUA levels over 7 mg/dl in men and over 6 mg/dl in women are considered high, although the parameter can vary considerably according to the geographical area and the ethnic group. Diabetes mellitus was defined as fasting blood sugar of ≥126 mg/dl or history of receiving treatment for diabetes mellitus or previously diagnosed diabetes mellitus. Metabolic syndrome was defined as per the NCEP ATP III guidelines.[10]

The patients having 3 or more of the parameters were classified as having metabolic syndrome: 1) Central obesity: waist circumference > 102 cm for male and > 88 cm for female; 2) Hypertriglyceridemia: Triglyceride ≥ 150 mg/dl or specific medications; 3) Low HDL cholesterol: < 40 mg/dl for male and < 50 mg/dl for female or specific medications; 4) Hypertension: Blood pressure ≥ 130 mm systolic or ≥ 85 mm diastolic or specific medications; 5) Fasting plasma glucose ≥ 100 mg/dl or specific medications or previously diagnosed type 2 diabetes mellitus.[11] A BMI (Body Mass Index) of ≥ 25 was taken as marker of being overweight. All patients were informed and the consent was obtained from all patients. Demographic data, including age, sex, and history of diseases like diabetes mellitus, hypertension, and Ischemic Heart Disease was recorded based on medical records of the patients. Smoking history and its duration were asked from the patients. Baseline blood pressure of the patients was recorded (in supine position). Patients were investigated for uric acid measured by the Uricase method,[12] FBG and postprandial blood glucose (PPBG), as described by the GOD-POD method (using glucose oxidase enzyme),[13] at admission and after 7 days i. e. at discharge. Additionally, the following laboratory investigations were performed as: S cholesterol

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(CH), estimated by cholesterol oxidase-phenol-aminophenazone (CHOD-PAP) method;[14] triglycerides (TG), using glycerolphosphate oxidase-phenol-aminophenazone (GPO-PAP) end point assay method;[15] high-density lipoprotein cholesterol (HDL-C), by polyethylene glycol cholesterol oxidase-phenol-aminophenazone (PEG-CHOD-PAP) method;[16] low-density lipoprotein cholesterol (LDL-C), as described by Bairaktar et al;[17] S ADA level, as described by Giusti and Galanti;[18] HbA1c, analyzed by a Nycocard Reader (Axis-Shield Diagnostics Ltd, Dundee, UK);[19] and body mass index (BMI).[20]

All the biochemical measurements were performed as per the standard procedures. Modified National Institute of Health (NIH) stroke scale score was calculated for all the patients of stroke at the time of admission and before discharge from hospital. The study protocol was approved by the Institutional Ethics Committee.

Exclusion Criteria: Subjects with following conditions were excluded from the study: 1) Patients with a known or emboli source (atrial fibrillation, valvular heart disease, patients receiving anticoagulant treatment; 2) Duration of symptoms more than 48 hours; 3) Past history of vascular disease (previous stroke, angina, myocardial infarction, revascularizations, peripheral artery disease); 4) Patients receiving drugs affecting SUA levels (diuretics, losartan, allopurinol, probenecid, atorvastatin, fenofibrate); 5) Active infections; 6) Malignancy; 7) Renal or liver disease; 8) Thyroid dysfunction; 9) Chronic obstructive pulmonary disease; 10) Chronic inflammatory bowel disease; 11) Excessive alcohol consumption 12) H/O chronic smoking.

STATISTICS: The data obtained were tabulated as a mean ± standard deviation (SD) and analyzed using Student's t-test. The level of significance was determined as its P value with P. 0.05 taken as not significant, P, 0.05 taken as significant at the 5% significance level, P, 0.01 taken as significant at the 1% significance level, and P, 0. 001 was taken as highly significant.

RESULTS: Out of 100 patients enrolled in the study, 69 were males and 31 were females. The Male: female ratio was 2.2: 1. The number of patients presenting in the group I was lower than group II i. e. 31 (31%) vs. 69 (69%). The difference between the age groups between two groups was not statistically significant (P = >0.05). Comparison of conventional risk factors for ischemic stroke (confounding variables) between cases and controls is shown in Table 1. The analysis of Table 1, show that the number of patients with hypertension, smoking, diabetes mellitus, metabolic syndrome, obesity and higher BMI was more in cases when compared with control.

The laboratory evaluations parameters are in both the groups are shown at admission [Table 2] and at discharge [Table 3]. Analysis of Table 2 &3 shows that Mean SUA level in group I and group II at admission was 5.09±1.07 & 6.48±1.92 mg/dl and at discharge was 4.85±7.07 & 6.28±1.92 mg/dl respectively. SUA level was significantly higher in group II, when compared with Group I both at admission and at discharge. Similarly, the FBS level at admission was 106±14.18 & 122.33±18.46 mg/dl and was 113±15.18 & 129.37±15.40 mg/dl at discharge in group I and group II respectively. FBS level was significantly higher in group II when compared with Group I both at admission and at discharge. HbA1c level at admission was 5.30±4.28 & 6.87±2.1 mg/dl at admission and was 5.30±4.28 & 6.87±2.10 mg/dl at discharge in group I and group II respectively. HbA1c level was significantly higher in group II, when compared with Group I both at admission and at discharge. Lipid profile at admission was as:

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- S. Triglyceride (mg/dl) at admission was 134.7±8.10 & 162.34±14.08 and was 139.7±18.1 & 154.34±10.12 mg/dl at discharge in group I and group II respectively. S. Triglyceride level was significantly higher in group II, when compared with Group I both at admission and at discharge. A significant positive correlation between SUA and serum triglycerides was also observed in the present study.
- S. Total Cholesterol (mg/dl) at admission was 146±14.07 & 169±12.10 and was 148±11.7 & 160.09±14.10 mg/dl at discharge in group I and group II respectively. S. Triglyceride level was significantly higher in group II, when compared with Group I both at admission and at discharge.
- S. HDL-C (mg/dl) at admission was 47.01±12.14 & 42.11±10.17 and was 49.12 ±9.14 & 45.16±11.40 mg/dl at discharge in group I and group II respectively. S. HDL-C was s significantly lower in group II, when compared with Group I both at admission and at discharge. Thus, there was a significant inverse correlation between SUA and HDL cholesterol.
- S.LDL-C (mg/dl) was 115.85±14.07 & 119.85±14.07 at admission and was 104.12±14.07 & 110.42±12.67 mg/dl at discharge in group I and group II respectively. There was a slight rise in S. LDL-C in group II, when compared with Group I both at admission and at discharge.

The S. ADA level at admission was 43.62±8.44 & 45.62±7.90 (U/L) at admission and was 45.62±11.20 & 49.47±9.57 (U/L) at discharge in group I and group II respectively. There was only a slight rise in S. ADA level in group II, when compared with Group I at admission and but this rise was significantly more at discharge.

DISCUSSION: The present study highlights the association between the increased serum uric acid levels and other risk factors with ischemic stroke. Hyperuriceamia was more commonly seen in patients presenting with ischemic stroke [Table 1]. Qin et al,[21] in a population-based on cross-sectional study in Shanghai in 8510 participants aged ≥40 years observed that high In the present study hyperuricemia was associated with high BMI, triglycerides, total cholesterol, LDL-C and low HDL-C. [Table II] Li et al,[22] in meta-analysis of 15 prospective studies with 22, 571 cases of stroke and 1, 042, 358 participants, observed that the presence of hyperuricemia was associated with greater risk of both stroke incidence (RR, 1. 22; 95% CI, 1.02-1.46) and mortality (RR, 1.33; 95% CI, 1.24-1.43). Since SUA level is a quantitative numerical variable, an increase in 1mg/dl has a 47. 0% (95% CI 1. 0% to 2.16%) increase in the odds ratio (OR) of having an ischemic stroke has been proposed by Khalil et al.[23]

The exact cause for this association is not known, but elevated uric acid might be injurious for large cerebral arteries with some probable confounding risk factors.[24] On the other hand De Leeum et al in European trial in patients with isolated systolic hypertension observed no significant relationship between serum uric acid levels with fatal and non-fatal strokes.[25] Mehrpour et al studied the role of uric acid as a risk factor for acute stroke and concluded that 47. 3% of the patients were hyperuricemic where as in present study 69% of the patients had increased uric acid. Hyperuricemia has been shown to be associated with increased amounts of triglycerides and Low-density lipoprotein (LDL) cholesterol,[26] and hyperuricemia was more commonly seen in men than women.[27,28]

Similarly, in present study hyperuricemia was seen to be twice as common in males as in females [Table. 1]. In this study, there was a significant association between serum uric acid level and diabetes mellitus, hypertension, high BMI and smoking [Table. 2 & 3]. Similarly, Li et al,[29] observed in cross-sectional study that hyperuricemia was associated with hypertension, insulin resistance, hyperglycemia, high BMI, hypercholesteremia, hyper-LDL-c, and hypertriglyceridemia. However, in a study by Bonora et al the association between smoking and hyperuricemia was not seen.[30]

The mechanism of this strong association between serum uric acid levels and triglyceride levels is not understood. Although some studies reported the role of genetic factors in the occurrence of gout and hypertriglyceridemia.[30] When S. ADA level was compared between group 1 and group 2, it was observed that there was a significant increase in S. ADA levels in group 2 (P> 0. 01) [Table 2 & 3]. Thus, it can be interpreted from the present study that hyperuricemia is associated with high adenosine deaminase (ADA) an enzyme involved in purine metabolism and needed for the breakdown of adenosine from food and for the turnover of nucleic acids in tissues. Similarly, it was observed in quail model supplemented high fat diet hyperuricemia was associated with hypercholesterolemia, high ADA and xanthine oxidase levels.[31]

CONCLUSION: Our study demonstrates that hyperuricemia in patients with acute stroke was significantly associated with population at risk. The Uric acid level was significantly higher in men than women presenting with ischemic stroke. Hyperuricemia was associated with ischemic stroke in patients with dyslipidemia, smoking, Diabetes mellitus, metabolic syndrome, hypertension and male sex and these factors can be considered as a risk factor for acute stroke. The finding of hyperuricemia in patients with ischemic stroke, suggests the clinical importance of monitoring and intervention based on SUA measurement, particularly because SUA is easily and routinely measured. In addition, control of blood sugar in diabetic patients, control of diet to manage hyperlipidemia, regular exercise to decrease weight & BMI and stoppage of smoking should be done to decrease onset of ischemic stroke.

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Parameter	Group I (n=31) (Control)	Group II (n=69) (Cases)
Age (years)	56.6 ± 9.98	55.9 ± 10.11^{NS}
Sex (Males)	20	46
Sex (Females)	11	23
Hypertension	12.43±5.67	15.7±7.02**
Diabetes mellitus	15.00±4.43	22±5.54**
Metabolic syndrome	18.25±2.62	28±7.68***
Smoking	14.21±2.69	19.1±2.61**
BMI > 25 (kg/m2)	21.70± 7.62	26.9±11.22**

Table 1: No of patients with Risk Factors for Ischemic Stroke Between two groups

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Values are expressed as mean ± standard deviation. NSP, >0.05 (not significant); *P, 0.05 (significant at 5% significance level); **P, 0.01 (significant at 1% significance level) and ***P, 0.01 (highly significant at 1% significance level). Abbreviations: ADA, adenosine deaminase; BMI, body mass index; FBS, Fasting blood sugar; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; S, serum.

Parameter	Group I	Group II
S Uric acid (mg/dl)	5.09 ± 1.07	6.48 ± 1.92**
FBS (mg/dl)	106± 14.18	122.33± 18.46**
HbA1c (mg/dl)	5.30± 4.28	6.87± 2.1***
S.Triglyceride (mg/dl)	134.7±8.10	162.34±14.08***
S.Total Cholesterol (mg/dl)	146± 14.07	169± 12.10**
S.HDL-C (mg/dl)	47.01 ±12.14	42.11 ± 10.17**
S.LDL (mg/dl)	112.85± 14.07	119.85± 14.07*
S.ADA (U/L)	43.62 ± 8.44	45.62 ± 7.90*

Table 2: Laboratory values of Ischemic Stroke Between two groups at admission

Values are expressed as mean ± standard deviation. NSP, >0.05 (not significant); *P, 0.05 (significant at 5% significance level); **P, 0.01 (significant at 1% significance level) and ***P, 0.01 (highly significant at 1% significance level). Abbreviations: ADA, adenosine deaminase; BMI, body mass index; FBS, Fasting blood sugar; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; S, serum.

Parameter	Group I	Group II
S Uric acid (mg/dl)	4.85± 7.07	6.28 ± 1.92**
FBS (mg/dl)	113± 15.18	129.37± 15.40**
HbA1c (mg/dl)	5.30± 4.28	6.82±6.21**
S.Triglyceride (mg/dl)	139.7±18.1	154.34±10.12**
S.Total Cholesterol (mg/dl)	148± 11.7	160.09± 14.10**
S.HDL-C (mg/dl)	49.12 ±9.14	45.16 ± 11.40**
S.LDL –C (mg/dl)	104.12± 14.07	110.42± 12.67*
S.ADA (U/L)	45.62 ± 11.20	49.47 ± 9.57**

Table 3: Laboratory values of Ischemic Stroke Between two groups at discharge

Notes: Values are expressed as mean ± standard deviation. NSP, >0.05 (not significant); *P< 0.05 (significant at 5% significance level); **>, 0.01 (significant at 1% significance level) and ***P> 0.001 (highly significant at 1% significance level). Abbreviations: ADA, adenosine deaminase; BMI, body mass index; FBS, Fasting blood sugar; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; S, serum.

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