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Original Research Article.

Correlation Between Serum Uric Acid and HbA1c in Patients of Type 2 Diabetes Mellitus

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ABSTRACT

Introduction: Diabetes Mellitus (DM) is a metabolic disorder. The prevalence of DM ranges from 0.4 – 3.9% in rural areas to 9.3 – 16.6% in urban areas in India. Hyperuricemia is defined as serum uric acid concentration in excess of urate solubility. Glycated Hemoglobin (HbA1c) is a very important biochemical parameter that provides long term status of blood glucose levels and serves as a monitoring tool for measuring glycemic control in Diabetic patients.

Aim: This study was designed to find the correlation between HbA1c and Serum Uric Acid in Type 2 Diabetes Mellitus patients.

Materials and Methods: The present study was a hospital based observational and analytical study. A total of 150 cases were divided into 2 groups (Group I – control group n= 50 and Group II – Diabetic patients n = 100 aged > 35 years) referred from The Department of Medicine, Rajindra Hospital, Patiala with Exclusion criteria (renal failure, renal transplant, hepatic disorders). Serum Uric Acid, HbA1c and Fasting Blood Glucose (FBS) were the parameters analysed.

Results: Serum Uric Acid, HbA1c, and FBS were found to be elevated. The study found a statistically significant positive correlation between Serum Uric Acid and HbA1c and FBS in Diabetic patients.

Conclusion: Serum Uric Acid may serve as a potential biomarker for monitoring glycemic control in patients of Diabetes Mellitus.

Keywords: HbA1c, Serum Uric Acid, Diabetes Mellitus.

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INTRODUCTION

Diabetes Mellitus (DM) is a metabolic disorder which is characterized by hyperglycemia and insufficiency of the secretion or the action of endogenous insulin. It is found in every population in the World and in all regions including rural parts of low- and middle-income countries. The number of people with Diabetes is steadily rising with WHO estimating there were 463 million adults with Diabetes Worldwide in 2019. International Diabetes Federation (IDF) estimates there will be atleast 629 million people living with Diabetes by 2045 and the global number of Diabetes cases might increase by 48% between 2017 and 2045.[1]

India is the diabetes capital with 69.1 million people with Diabetes Mellitus, second highest number of cases after China in 2015.[2] Prevalence of diabetes is higher in the Indian Subcontinent and it is rapidly rising at an alarming rate. Over the past 30 years, the status of Diabetes has changed from being considered as a mild disorder of the elderly to one of the major causes of morbidity and mortality affecting the youth and the middle-aged people.[3]

HbA1c represents the average blood glucose level within the past 3 months. It develops when hemoglobin, a protein within RBC that carries O_2 throughout body, joins with glucose in the blood and becomes glycated.[4] HbA1c levels could be used as an independent risk factor for stroke and CVD in both healthy and Diabetic persons.[5,6] Uric acid is an end product of the purine metabolism.[6] Hyperuricemia is associated with an increased future risk of diabetes due to its relationship with metabolism of glucose.[7-12] Hyperuricemia plays a role in the development of metabolic syndrome. During metabolic syndrome, resistance to insulin which is at the center of its pathophysiology could originate from the oxidative stress generated by Hyperuricemia.[8] The participation of Hyperuricemia in the genesis of the metabolic syndrome suggests that it may participate in poor glycemic control during diabetes. The aim of the present study was to study the possibility of any correlation of HbA1c and serum uric acid in Type 2 Diabetes Mellitus patients.

MATERIALS AND METHODS

The present hospital based observational and analytical study was conducted in the department of Biochemistry, GMC, Patiala, Punjab, India in Collaboration with department of Medicine, Rajindra Hospital, Patiala. Patients having DM were selected for the present study. An informed consent was taken from every patient. The project was approved by the Ethical Committee of the institute. The present study comprised of 150 individuals, divided into following groups:

1. Group I: Control Group – n = 50 (normal healthy subjects > 35 years)
2. Group II: Study Group – n = 100 (type 2 DM subjects > 35 years irrespective of their glycemic control and duration of diabetes).

Exclusion Criteria: Patients having renal failure, renal transplant, long term use of diuretics and steroids, hepatic disorders and regularly consuming alcohol were excluded from the study.

Fasting blood samples were collected under aseptic conditions and were allowed to clot. Serum/ plasma was separated and analyzed for Fasting blood sugar and serum uric acid. For HbA1c, sample was collected in EDTA vacutainers.

1. Serum uric acid was estimated by uricase- Peroxidase methods as described by Balis ME 1976.[9]
2. The Glycosylated Hb (HbA1C) was estimated by Ion-Exchange resin method as described by Trivelli et al 1971.[11]
3. Fasting blood sugar was estimated by Glucose – Oxidase method as described by Trinder P 1969.[10]

The tests were performed by using commercially available kits from Transasia Biomedicals.

Statistical Analysis

The data collected was analyzed using computer software SPSS version 23.0. Student's t-test was applied to study the variation between two groups. Pearson's Coefficient of correlation was calculated to study the significance of correlation between different parameters. The level of significance was used p value < 0.05.

Table 1: Comparison of age in control and study group

Group	Age (mean ± SD)
Group –I (Control)	48.88 ± 10.42
Group – II (Study)	56.55± 10.05***

*** p< 0.001 Age was comparable

Table II: Biochemical parameters in control & study group

Parameter	Control group (mean ± SD)	Study group (mean ± SD)	P - Value
Serum Uric Acid (mg%) (Normal range = 2 – 7 mg%)	4.34 ± 0.66	8.41 ± 1.19	0.05
Fasting Blood Sugar (mg%) (Normal range = 60 – 110 mg%)	74.90 ± 10.13	190.37 ± 21.08	0.05
HbA1c (%) (Good control < 7%)	5.62 ± 1.19	7.91 ± 1.00	0.001

All parameters were increased in the study group.

Table III: Correlation of HbA1c with different biochemical parameters

Parameter	Correlation Coefficient (r)	P – value
Serum uric acid	+ 0.10	0.041
Fasting Blood Sugar	+ 0.43	0.001

*HbA1c is having positive correlation with serum uric acid (+0.10) and Fasting blood sugar (+0.43).

RESULTS AND DISCUSSION

The present study was focussed on evaluation of HbA1c, serum uric acid in diabetes mellitus patients and comparing their results with healthy individuals (controls). In the study group patients had a mean age of 56.55 ± 10.05 and in control group the mean age was 48.88 ± 10.42.

According to the observed outcomes, the HbA1c levels were significantly increased in study group (7.91 ± 1.00) (p < 0.001) as compared to the control group (5.62 ± 1.19). The serum uric acid levels were significantly increased in cases (8.41 ± 1.09) (p < 0.001) as compared to control group (4.34 ± 0.66). Fasting blood sugar of patients was 190.37 ± 21.08 (p < 0.001) which was significantly higher than control group 74.90 ± 10.13.

The correlation of HbA1c with serum uric acid was positive (r = + 0.10, p = 0.041) and statistically significant. It may be due to the relationship between elevated serum uric acid and HbA1c in diabetic mellitus patients linked to the defective reabsorption of uric acid in the proximal tubules in diabetic individuals. An elevated level of uric acid is a risk factor for peripheral arterial disease, insulin resistance and components of the metabolic syndrome.[13]

The association between the high serum uric acid and glycated hemoglobin during type2 DM has often described in other epidemiological studies.[12,14,22] Excessive uric acid will lead to an increase in reactive oxygen species (ROS) production, which

leads to inflammation and dysfunction in the blood vessels. Uric acid mediated oxidative stress induced lipid peroxidation, DNA damage and activation of inflammatory factors finally leads to cellular damage.[15,16] Oxidative stress also can affect the expression of insulin gene, causing a decrease in insulin secretion. The reason for the increase in serum UA levels associated with obesity and metabolic dysfunction is not exactly known, but several mechanisms could be responsible, including the following: 1) increased intake of dietary purines, alcohol, and fructose, which produce UA.[17,18]; 2) impaired renal function and renal microvascular disease, which can increase UA production and/or decrease UA clearance[19]; and 3) hyperinsulinemia, which increases renal UA reabsorption.[20]

A statistically significant positive correlation was observed in HbA1c and FBS ($r = +0.43$, $p < 0.05$). HbA1c is commonly used as clinical indicator for glycemic control. It is an indicator of average blood glucose concentration over the period of 2 – 3 months[15] and it is the gold standard to monitor diabetic control. It is also emerging as the first line of screening test for diagnosing diabetes mellitus.[15,8]

CONCLUSION

High serum uric acid levels have been observed in our study. Our results have shown a positive correlation between serum uric acid levels and HbA1c levels in diabetic patients. All these parameters were found to be elevated. This study indicates the importance of screening these parameters in patients of DM. It is possible that an increase in serum uric acid concentration is a protective mechanism to attenuate the adverse effects of an increase oxidative stress. Raised serum uric acid levels have an adverse impact on glycemic control. Thus, estimation of serum uric acid may serve as potential biomarker for monitoring the glycemic control in patients of Diabetes mellitus. Further studies are needed to evaluate the effect of serum uric acid lowering drugs on the glycemic control in patients of Diabetes Mellitus.

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