

A study on correlation of serum uric acid level and glycemic control among patients of type II Diabetes Mellitus at a tertiary care center of Bihar

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Abstract

Introduction: Diabetes mellitus is a metabolic disorder which is characterized by hyperglycaemia and insufficiency of the secretion or the action of endogenous insulin. Although the aetiology of the disease has not been well defined, viral infections, autoimmune diseases, and environmental factors have been implicated. With this background, the present study was undertaken to find the correlation of the serum uric acid and the glycemic control in patients with type 2 Diabetes mellitus. The proxy indicator used for glycemic control here was glycated hemoglobin level (HbA1C). **Methodology:** The present case control study was undertaken in the Department of Physiology of J.N.K.T. Medical College, Madhepura, Bihar, India. The study period was 6 months that was from May 2020 to October 2020. Prior ethical approval was obtained from the Institutional ethics committee. The subjects who were included in this study were divided into 2 groups: Group A included 20 normal healthy individuals, in the age group 40-65 years, of either sex and without any family history of DM. Group B included 20 diagnosed patients of Type 2 Non-Insulin Dependent Diabetes mellitus (NIDDM), who were in the age group 40-65 years, of either sex, from the same population. **Results:** The mean age of the patients in group A was 31.8 ± 7.9 years and that of patients in group B was 33.6 ± 6.8 years. In the control group, there was a slight male predominance with 55% being male, while in the study group, female dominant with 75%. There was no statistical difference between both the groups based on gender and age. The mean FBS level of group A was 76.2 ± 8.3 mg/dl and that of group B was 144.9 ± 62.4 mg/dl, which was significantly higher than that of group A ($p < 0.05$). **Conclusion:** In conclusion, our study suggests that there is an increase in the serum uric acid levels with an increase in the HbA1C levels. This showed a positive relationship in Type 2 Diabetes mellitus.

Key Words: correlation, serum uric acid, glycemic control, type II Diabetes Mellitus

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Introduction

Diabetes mellitus is a metabolic disorder which is characterized by hyperglycaemia and insufficiency of the secretion or the action of endogenous insulin. Although the aetiology of the disease has not been well defined, viral infections, autoimmune diseases, and environmental factors have been implicated[1]. The prevalence of diabetes has been growing rapidly from 135 million in 1995 to an estimated 380 million in 2025[2]. Insulin, a hormone that regulates the body's use of glucose (blood sugar), is released by the cells of the pancreas which are called the islets of Langerhans. If the pancreas malfunctions, it may produce an inadequate supply of insulin, or no insulin at all. Type 1 diabetes mellitus then develops[3].

Type 2 diabetes mellitus is a heterogeneous disease which is characterized by variable degrees of insulin resistance, impaired insulin secretion and increased glucose production. Insulin resistance occurs when the cells become less sensitive to the effects of insulin[4]. Uric acid is the end product of the purine metabolism in humans[5]. Although hyperuricaemia and gout are associated with an increased future risk of diabetes, diabetes may reduce the future risk of gout through the uricosuric effect of glucose or the impaired inflammatory response[6]. The recognition of high levels of serum uric acid as a risk factor for diabetes has been a matter of debate for a few decades, since hyperuricaemia has been presumed to be a consequence of the insulin resistance rather than its precursor[7]. With this background, the present study was undertaken to find the correlation of the serum uric acid and the glycemic control in patients

with type 2 Diabetes mellitus. The proxy indicator used for glycemic control here was glycated hemoglobin level (HbA1C).

Methodology

The present case control study was undertaken in the Department of Physiology of J.N.K.T. Medical College, Madhepura, Bihar, India. The study period was 6 months that was from May 2020 to October 2020. Prior ethical approval was obtained from the Institutional ethics committee.

The subjects who were included in this study were divided into 2 groups: Group A included 20 normal healthy individuals, in the age group 40-65 years, of either sex and without any family history of DM. Group B included 20 diagnosed patients of Type 2 Non-Insulin Dependent Diabetes mellitus (NIDDM), who were in the age group 40-65 years, of either sex, from the same population. Informed consents were taken from all the subjects who were included in the study. The patients who suffered from Type 1 Diabetes mellitus, those with the acute complications of Diabetes mellitus, those with a history of acute infections and other ailments like gross congestive heart failure, tuberculosis, gout, rheumatoid arthritis and skeletal muscle injury, those with serum creatinine levels of >1.5 mg/dl and renal failure were excluded from the study.

A detailed history was taken from each patient and a thorough clinical examination was carried out on each patient. Fasting blood samples were drawn and they were investigated for uric acid, blood sugar and HbA1C, and the values were compared with those of normal healthy subjects. Serum uric acid was estimated by an Enzymatic colorimetric method[8], Fasting blood was estimated by an Enzymatic colorimetric method[8], Fasting blood sugar (FBS) was estimated by the GOD-POD Method[9] and HbA1c was estimated by using a Nycocard Reader[10].

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The comparisons were done by using the Student's 't' test on the number of variables for each parameter. The correlations were done by Pearson's correlation analysis.

Results

The mean age of the patients in group A was 31.8 ± 7.9 years and that of patients in group B was 33.6 ± 6.8 years. In the control group, there was a slight male predominance with 55% being male, while in the study group, female dominant with 75%. There was no statistical difference between both the groups based on gender and age. The mean FBS level of group A was 76.2 ± 8.3 mg/dl and that of group B

was 144.9 ± 62.4 mg/dl, which was significantly higher than that of group A ($p < 0.05$). In the present study, it was observed that the HbA1C value of group B was statistically significant higher than that of group A, the value of group A being $4.2 \pm 0.6\%$ and that of group B being $8.1 \pm 2.1\%$ ($p < 0.05$). Similar findings were observed in the values of the serum uric acid levels, which showed a significantly higher value for group B as compared to that of group A ($p < 0.05$). Mean serum uric acid level of group A and B patients were 5.2 ± 1.1 mg/dl and 6.9 ± 1.5 mg/dl. The Pearson's correlation co-efficient for the correlation between serum uric acid and HbA1C showed a positive correlation with a Pearson's coefficient of 0.62 [Table 1].

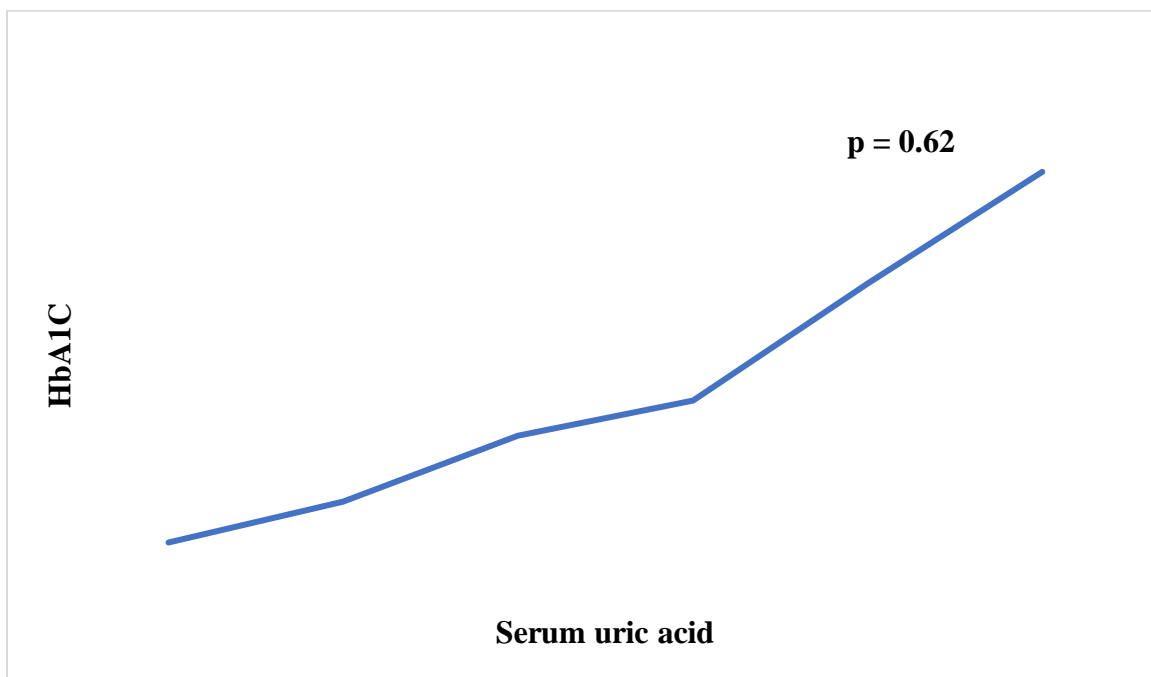


Fig 1: A line diagram showing relation of serum uric acid levels and HbA1C levels of all the 40 participants

Discussion

Diabetes mellitus is the most common endocrinological disorder which is characterized by metabolic abnormalities and long term complications[11]. The prevalence of diabetes has been growing rapidly from 135 million in 1995 to an estimated 380 million in 2025[2]. Type 2 Diabetes mellitus or non-insulin dependent Diabetes mellitus (NIDDM) typically occurs in old age and in obese people[12]. Type 2 Diabetes mellitus is a heterogeneous disease which is characterized by variable degrees of insulin resistance, impaired insulin secretion and increased glucose production[4]. In the present study, it was observed that the serum uric acid level of group B was significantly higher than that of Group A ($p < 0.001$). The serum uric acid levels were increased in the Type 2 diabetic patients and they were associated with the insulin resistance syndrome, impaired glucose tolerance and nephropathy. The clearance of uric acid gets reduced, with an increase in the insulin resistance. The actual mechanism of hyperuricaemia, which was found in these patients, was not known, but it was observed that the compensatory hyperinsulinaemia in the insulin resistant individuals imposed an antiuricosuric effect on the kidneys[13]. Similar results were reported by Joseph B. Herman et al., [14], T P Whitehead et al.[15], and Causevic et al.,[16].

There was a significant ($p < 0.05$) positive correlation ($r = 0.092$) between serum uric acid and HbA1C, which meant that there was an increase in the serum uric acid with an increase in HbA1C. This can be explained on the basis of the mechanisms which suggest an association of hyperinsulinaemia with increased uric acid production. An increased purine biosynthesis which occurs due to an increased

activity of the hexose monophosphate pathway shunt[17] can be conceptually linked to the disorders which are characterized by insulin resistance and/or hyperinsulinaemia. The increased flux of glucose-6-phosphate through the hexose monophosphate pathway shunt due to impairment of the glycolytic pathway, has been suggested as an explanation for the increased uric acid in impaired glucose tolerance[14] and this may also include excess carbohydrates and an enhanced lipogenesis in the presence of excess insulin[18]. Similar findings were explained by HK Choi et al.,[19].

Conclusion

In conclusion, our study suggests that there is an increase in the serum uric acid levels with an increase in the HbA1C levels. This showed a positive relationship in Type 2 Diabetes mellitus.

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