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"Correlation between Serum Uric Acid Level and Glycated Haemoglobin Level in Patients of Diabetes Mellitus A Hospital Based Study"

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Abstract:
1. Background: Over the past three decades, the number of people with diabetes mellitus has increasing rapidly worldwide and it is one of the most important public health challenges to all country. Therefore, the present study was undertaken to compare the levels of HbA1c, serum uric acid in patients with newly diagnosed diabetes mellitus with normal healthy individuals.
2. Material and Method: This is study was undertaken in the Department of Lab Medicine, RIMS Ranchi, and Jharkhand India. Total 300 subjects included in this study (Group I: 150 normal healthy individuals, who were in the age group 30-80 years, of either sex. Group II: 150 newly diagnosed patients of diabetes mellitus in the same age group). Fasting blood samples were taken and investigated for blood sugar, HbA1c and serum uric acid and value compared with those of normal healthy subjects. Means ± standard deviation were calculated and student t-test was applied to find out significance level.
3. Results: Mean serum levels of FBS (89.71 ± 9.22), HbA1c (4.68 ± 0.47), uric acid (4.21 ± 1.04) in (group-I control group) and FBS (145.57 ± 76.61), HbA1c (8.94 ± 2.38), uric acid (7.01 ± 0.27) in (group-II).
4. Conclusion: Increased serum uric acid was seen with reduced glucose tolerance hence early estimation of both the parameters should be done while monitoring case of diabetes and thus will help to decrease the incidence of renal complications.
Key Words –*Diabetes Mellitus, Serum Uric Acid, Glycated Haemoglobin.*

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I. 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].

Hyperuricaemia is considered the precursor of gout, which is the most common inflammatory arthritis in adult men[8]. Several studies have shown that a moderate degree of hyperglycaemia is associated with higher serum uric acid levels, while a higher degree of hyperglycaemia (>10 mmol/l [180 mg/dl]) is associated with lower serum uric acid levels[9,10,11,12]. This bell-shaped relationship raises an interesting implication of the diverging risk of hyperuricaemia or gout among pre-diabetic vs diabetic individuals.

The present study was undertaken to compare the levels of HbA1c and serum uric acid in patients with newly diagnosed diabetes mellitus with normal healthy individuals.

II. Materials And Methodology

This is a cross sectional study and was undertaken in the Department of Lab Medicine, RIMS Ranchi, and Jharkhand India. Total 300 subjects included in this study

- Group I: 150 normal healthy individuals, who were in the age group 30-80 years, of either sex.
- Group II: 150 newly diagnosed patients of diabetes mellitus in the same age group). Means ± standard deviation were calculated and student t-test was applied to find out significance level.

Informed and written consents were taken from all the subjects who were included in 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 serum uric acid, blood sugar and HbA1C, and the values were compared with those of normal healthy subjects. Fasting blood samples were drawn and investigated for uric acid, blood sugar and HbA1c and were analysed on dirui and bio -rad analyzers.

III. Results

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. A logistic regression analysis was also done on the variables of each parameter.
The statistical analysis showed no difference between the sexes with regards to their mean age. The sex and the number distribution in these groups were comparable

Table – I: Showing the comparison in the gender between the two groups.

Sex	Group – I	Group – II	Total
Male	78 (52%)	60 (40%)	138 (46%)
Female	72 (48%)	90 (60%)	162 (54%)
Total	150	150	300

Mean serum levels of FBS, HbA1c and uric acid were 89.71 ± 9.22, 4.68 ± 0.47 and 4.21 ± 1.04 in controls (group-I) & 145.57 ± 76.61, 8.94 ± 2.38 and 7.01 ± 0.27 in cases (group-II) respectively. The results indicate there is a statistically significant difference between the two groups.

Table – II: Depicting Mean ± Standard deviation and significance of different parameters between two groups.

Parameters	Group – I	Group – II	p-value
Age in years	55.72 ± 12.92	55.42 ± 12.77	>0.05
FBS mg/dL	89.71 ± 9.22	145.57 ± 76.61	<0.001
HbA1c %	4.68 ± 0.47	8.94 ± 2.38	<0.001
Serum Uric acid mg/dL	4.21 ± 1.04	7.01 ± 0.27	<0.001

IV. Discussion

In the present study, it was observed that the FBS levels of cases were significantly higher than that of controls. In our study the mean HbA1c levels for controls is 4.68 ± 0.47 and for diabetic cases is 8.94 ± 2.38. HbA1c levels are significantly raised in newly diagnosed Type 2 diabetic cases when compared with the controls.

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-value) 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. Similar results were reported by Joseph B. Herman et al., T P Whitehead et al., and Causevic et al.

There was a significant positive correlation 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 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 and this may also include excess carbohydrates and an enhanced lipogenesis in the presence of excess insulin.

V. Conclusion

In conclusion, our study suggests that there is Increased serum uric acid was seen with reduced glucose tolerance hence early estimation of both the parameters should be done while monitoring case of Type-2 diabetes and thus will help to decrease the incidence of renal complications.

References

[1]. Maritim AC, Sanders RA, Watkins JB. Diabetes, Oxidative Stress, and Antioxidants: A Review Journal Biochem Molecular Toxicology. 2003; 17(1): 24-28.
[2]. Singh PP, Mahandi F, Roy A, Sharma P. Reactive oxygen Species, reactive nitrogen species and oxidants in etiopathogenesis of Diabetes Mellitus Type-2. Indian Journal of Clinical Biochemistry. 2009; 24(4): 324-42.
[3]. "Defination of c-peptide". http:// www. medicinenet .com.
[4]. Soliman GZA. Blood lipid peroxidation (Superoxide dismutase, malondialdehyde, glutathione) levels in Egyptian type 2 diabetic patients. Singapore Med Journal. 2008; 49(2): 129-36.
[5]. Safi AJ, Mahmood R, Khan MA, Ami-ul-Haq. Association of serum uric acid with type II Diabetes Mellitus. JPMI. 2002; 18(1): 59-63
[6]. Luis A.G.R, Lucia C.S, Choi H.K. Impact of diabetes against the future risk of developing gout. Journal For Health Professionals and Researches In Rheumatic Diseases. 2010 Dec; 69(12): 2090-94.
[7]. Butler R, Morris AD, Belch JJ, Hill A, Struthers AD. Allopurinol normalizes endothelial dysfunction in type 2 diabetics with mild hypertension. Hypertension Journal of American Heart Association. 2000; 35: 746- 51.
[8]. Roubenoff R, Klag MJ, Mead LA, Liang KY, Seidler AJ, Hochberg MC. Incidence and risk factors for gout in white men. J Am Med Assoc 1991;266:3004–7.
[9]. Cook DG, Shaper AG, Thelle DS, Whitehead TP. Serum uric acid, serum glucose and diabetes: relationships in a population study. Postgrad Med J 1986;62:1001–6
[10]. Tuomilehto J, Zimmet P, Wolf E, Taylor R, Ram P, King H. Plasma uric acid level and its association with diabetes mellitus and some biologic parameters in a biracial population of Fiji. Am J Epidemiol 1988;127:321–36.
[11]. Herman JB, Goldbourt U. Uric acid and diabetes: observations in a population study. Lancet 1982;2:240–3.
[12]. Whitehead TP, Jungner I, Robinson D, Kolar W, Pearl A, Hale A. Serum urate, serum glucose and diabetes. Ann Clin Biochem 1992;29:159–61.

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