

# A study of uric acid in diabetic patients

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## Abstract

**Aim:** To study the effect of DM on serum uric acid level. **Materials and Methods:** The present study was carried out in Department of Biochemistry, GMCH, Nagpur from May 2003 to May 2005, selected from diabetic OPD, GMCH, Nagpur in the age groups of 15 to 80 years. In all 30 controls, 30 type I DM and 30 type II DM patients were selected. The serum thus obtained was used for uric acid estimation using uricase method. **Results:** The present study showed that there was significant increase in uric acid levels in type I and type II DM as compared to healthy controls. **Conclusion:** Increased uric acid in DM suggests that DM has significant effect on purine metabolism.

**Keywords:** Type I DM, Type II DM, Uricase method.

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## INTRODUCTION

Diabetes Mellitus (DM) is a chronic disorder characterized by impaired metabolism of glucose and other energy yielding fuels as well as by late development of vascular and neuropathic complications.<sup>1</sup> Uric Acid<sup>2</sup> is the chief end product of purine catabolism in humans. It is synthesized in liver from where it is carried to kidneys. Normally 500-600 mg Uric acid is synthesized. Not all, is excreted in urine, some uric acid is excreted in bile. About 1130 mg of uric acid is present as miscible pool. Hyperuricemia<sup>3</sup> occurs either due to increased production of uric acid or due to decrease uric acid excretion or due to both. The causes of hyperuricemia due to increased production of uric acid are: HGPRT ase deficiency, PRPP synthetase overactivity, hemolytic process, lymphoproliferative disease, myeloproliferative disease, polycythemia vera, psoriasis, pagets disease, glycogenosis (III, V, VII) rhabdomyolysis, purine rich diet, obesity, Alcoholism. The causes of hyperuricemia due to decreased uric acid excretion are renal insufficiency, polycystic kidney disease, diabetes

insipidus, hypertension, starvation, hyperparathyroidism, toxemia of pregnancy, lead intoxication, down syndrome, hyperthyroidism, drugs (salicylates, diuretic, alcohol, levodopa, ethambutol, nicotinic acid, pyrazinamide. Uric Acid can cause  $\beta$ -cell necrosis, which produces a diabetogenic effect.

## MATERIAL AND METHODS

The present study was carried out in Department of Biochemistry, GMCH, Nagpur from May 2003 to May 2005. Cases were selected from amongst the patients attending diabetic OPD of GMC, Nagpur, under Department of Medicine. We selected 60 patients diagnosed as having DM from above mentioned patients population. The patients were aged between 15 to 80 years. A case of DM was considered to be eligible for inclusion in present study, only when the following criteria were being fulfilled. These criteria are 1) Patient attending DM OPD of GMCH. 2) Patient willing to enter study. 3) Patient with no h/o of hypertension, TB, smoking and alcohol. We selected 30 healthy, normal volunteers with ages ranging from 15-80 years as controls. Ethical clearance was obtained from institutional ethics committee. About 5 ml of fasting<sup>4</sup> venous blood was withdrawn from each control/patient using a disposable syringe and needle and under all aseptic precautions. The blood obtained thus was collected in a sterile bulb and allowed to clot at room temperature for at least 20 minutes. After this serum was separated by centrifugation. The serum thus obtained was used for the following estimations without further delay. All the water used in following estimation was distilled and deionized and all reagents used were of analytical grade. Estimation

of uric acid was done colorimetrically using uricase method.<sup>5,6</sup>

### STATISTICAL ANALYSIS

Data was analyzed on statistical software Intercostal stata version 7.0. Continuous variables are presented as Mean ± SD (Standard Deviation). Comparison between variables was done by using student-t-test. Analysis of variance (ANOVA) was used to see significant difference between variables. Categorical variables are represented in percentages. Categorical data was analyzed by using Chi-square-test and p<0.05 was considered as statistically significant.

### OBSERVATIONS AND RESULTS

**Table 1:** Distribution of Study subjects according to sex

	Controls	%	Type I DM	%	Type II DM	%
Male	10	33.33	10	33.33	10	33.33
Female	20	66.66	20	66.66	20	66.66
<b>Total</b>	<b>30</b>	<b>100</b>	<b>30</b>	<b>100</b>	<b>30</b>	<b>100</b>

Table: 1 shows the distribution of male and female patients. It was found that there were 33.33% males and 66.66% females in type I and type II DM whereas, control subjects were also found to be of same percentage e.g. 33.33% males and 66.66% of females.

**Table 2:** Uric Acid levels in sera of controls, Type I DM and Type II DM patients

Sr. No.	Study Group	Mean ± S.D.	Range of values
1	Control	5.25 ± 1.61	2.5 – 7.5
2	Type I DM	15.52 ± 2.01 * p < 0.001	8.2 – 21.5
3	Type II DM	13.02 ± 3.19 ** p < 0.001	7.2 – 15.82

Uric Acid values are expressed as mg/dl and normal range as given in Tecordiagnostic kit 2-7 mg/dl.

\*Gp I Vs Gp II Highly significant change.

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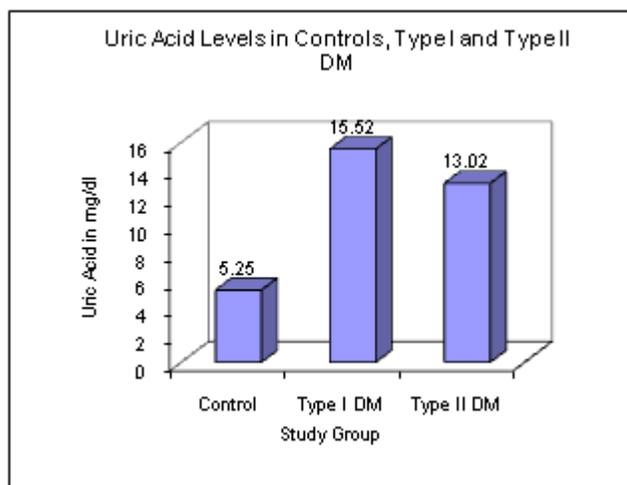


Table 2 depict the content of uric acid levels in controls type I DM and type II DM. It was observed that there was significant increase in uric acid level in type I and II DM as compared to controls.

### DISCUSSION

In the present study we found significant increase in serum uric acid level in Type I and Type II DM as compared to controls. Our reports support the finding of M Modan *et al*<sup>7</sup>, who studied the relationship between serum uric acid, DM and obesity. They suggested that the elevated serum uric acid may be associated with increased risk of atherosclerotic heart disease, hyperinsulinemia and insulin resistance. Tae Woo Yoo *et al*<sup>8</sup> studied the relationship between serum uric acid concentration and insulin resistance and metabolic syndrome. They concluded that as serum uric acid concentration increases the risk of metabolic syndrome and insulin resistance increases. Gluseppe Seghieri *et al*<sup>9</sup> found out relationship between serum uric acid level and patients with type II DM. They found out that serum uric acid level was significantly higher in patients of type II DM patient as compared to controls. Jesper O clauses *et al*<sup>10</sup> analyzed the relationship between fasting serum uric acid and insulin sensitivity index in a population based sample of 380 young healthy caucasian. They concluded that hypertriglyceridemia is a biochemical feature of insulin resistance syndrome. Hence hyperuricemia appears to be an indirect part of insulin resistance syndrome through its association with hypertriglyceridemia. M Gulfith<sup>11</sup> suggested the mechanism of diabetogenic action of uric acid by showing β cell lesion. In view of our findings in the present study it may be stated that determination of uric acid levels enabled us to conclude that above said parameter can be of valuable diabetogenic factor which result in abnormal glucose tolerance in addition to clinical picture and haematological investigations.

### REFERENCES

1. Cecil, Textbook of Medicin. 22<sup>nd</sup> Edition Vol. 2, Page 1424.
2. Textbook of Medical Biochemistry, 5<sup>th</sup> Edition, M N Chatterjee and Rama Shinde, Page 210, 211.
3. Harrison’s Principles of internal medicine, 15<sup>th</sup> Edition Vol. 2, Page 2269.
4. C A Burtis, E R Ashwood, D S Young, E W Bernes; Teitz’s Fundamentals of clinical chemistry- Chapter 2- Specimen Collection and other preanalytical variables, WB Saunders and Co. Ltd. 5<sup>th</sup> ed., 2003; 1:30-54.
5. Morin L G; Determination of serum urate by direct acid Fe<sup>3+</sup> reduction or by absorbance change (at 293 nm) on oxidation of urate with Alkaline Ferricyanide. Clin Cham, 1974; 20(1): 51-78.

6. Pierof F, Lorenzo P; Use of 3-5-Dichloro-2-hydroxybenzenesulphonicacid/4-amino-phenazone chromogenic system in direct enzyme assay of uric acid in serum and urine. Clin Chem. 1980; 26(2): 227.
7. Modan M, Halkin H, Karasik A and Lusky A; Elenated serum uric acid facet of hyperinsulinemia Diabetologin 1987; 30: 773-718.
8. Yoo T W, Sung K C, Shin H S, Kim B J, Kim B S, Kang J K, Lee M H, Park J R, Kim H, Rhee E J, Lee W Y, Kim S W, Rjn S H, Kerm D G; Relationship between serum uric acid concentration and insulin resistance and metabolic syndrome, Circ J 2005; 6 : 928-33.
9. Seghieri G, Moruzzo D, Stefano F, Banbini C, Anichini R, Alessandra D B, Lorenz O A, Flania F; Increase in serum uric acid is selectively associated with stroke in type II DM, Diabetes Care 2002; 25(6) : 1095.
10. Jesper O C, Kurt Borch-Johnson, Haus I, Oluf P; Analysis of the relationship between fasting serum uric acid and the insulin sensitivity index in a population based sample of 350 young healthy concasions. European journal of endocrinology, 1998; 138: 63-69.
11. Gillish M; The mechanism of the diabetogenic action of uric acid, J Biol Chan 1950; 84(1): 289-88.

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