

**Original Research Article**

A Study on the Correlation of Serum uric acid and Dyslipidemia with Glycaemic Status in Type 2 Diabetes Mellitus

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Abstract

Objectives: Our study was to detect the correlation of serum uric acid level with glycaemic status and with lipid profile. And also evaluate the various biochemical parameters, anthropometric measurements, blood pressure, serum uric acid level and associated factors.

Methodology: A 100 subjects with type 2 diabetes mellitus as a case and 100 subjects with non diabetics as control with age group greater than 40 years were enrolled in this study. A detail history, dietary pattern, clinical examination and relevant investigation were performed. Anthropometric examination like as measurement of BMI, measurement of waist-hip ratio and biochemical investigations like as blood glucose, Serum HbA1C estimation, Serum uric acid and Serum lipid profile were performed to all subjects.

Results: Data was analyzed by using SPSS software (Version 17). Mean \pm SD was observed. One way analysis of variance (ANOVA) with post hoc analysis using Tukey's multiple comparison test and Pearson correlation coefficient (r) was applied. P value was taken ≤ 0.05 for significant differences.

Conclusions: Type 2 diabetes mellitus patients is a strong negative correlation between blood glucose level and serum uric acid level. So that serum uric acid can be used as an important parameter to assess future cardiovascular risk in a type2 diabetes mellitus patient.

Keywords: Type 2 diabetes mellitus, anthropometric examination, Serum uric acid, serum lipid profile

Introduction

Diabetes mellitus (DM) is a hereditary, chronic and endocrine metabolic disorder.^[1] It may be associated with a number of complications including microangiopathies e.g. nephropathy, neuropathy, retinopathy, dermopathy and macroangiopathies e.g. coronary artery disease (CAD), cerebrovascular disease, peripheral vascular disease.

India, a developing Asian country with fast industrialization and a modern lifestyle is facing a grave problem in having the largest number of people with diabetes^[2,3] which is estimated to reach 80 million by the year 2030.^[4,5] It is close to becoming the diabetic capital of the world. The age of the diabetic patients play a significant role in the risk of developing type 2 DM especially after 40yrs.^[6] Type 2 DM is caused by relatively

impaired insulin secretion and peripheral insulin resistance.^[7,8] Lack of insulin or relatively low insulin levels affects the metabolism of carbohydrate, protein, fat, water and electrolyte balance resulting in diabetes.^[9]

Several distinct types of diabetes mellitus exists and are caused by a complex interaction of genetics, environmental factors and most importantly on lifestyles. There is a very important role of diet on both causation and treatment of diabetes mellitus. Depending on the etiology of the diabetes mellitus factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose utilization and increased glucose production. The metabolic dysregulation associated with diabetes mellitus causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes as well as on the health care system. The incidence of cardiovascular disease is increased in individuals with type 2 diabetes mellitus. The Framingham Heart study revealed a marked increase in coronary artery disease, myocardial infarction and sudden cardiac death in diabetes mellitus patients. The absence of chest pain (silent ischaemia) is also very common in patients of diabetes mellitus. So, to avoid such catastrophies various biochemical blood parameters, cardiological investigations should be done as a part of follow up in a diabetes mellitus patients.

Plasma uric acid, an end product of purine metabolism, is related to the purine bases of the nucleic acids. Its levels are genetically determined, but are influenced by multiple environmental factors. Previously it had been thought to be a metabolically inert end product without any physiological significance. Recently, it has been shown that there is a definite relationship between hyperglycemia and uric acid levels. Studies done so far have shown that, in the early stages of diabetes, the levels were high and as the diabetic status progresses, there is a gradual decline of uric acid levels in many patients. Studies showed that uric acid can act as an

important water soluble antioxidant.^[10,11] Urate, the soluble form of uric acid, can scavenge the superoxide and the hydroxyl radical and it also can chelate the transition metals.^[12] In a study by J. Fang, M.H. Alderman on serum uric acid and cardiovascular mortality it has been shown that serum uric acid level has a continuous, independent, specific and significant negative relationship with cardiovascular mortality.^[13] Studies have shown that serum uric acid level is negatively correlated with serum total cholesterol, LDL-cholesterol and triglyceride level, and positively correlated with serum HDL-cholesterol level. This dyslipidemia is also a cause of cardiovascular mortality.^[14]

There are evidences to suggest that low serum uric acid levels may precede the onset of diabetic retinopathy. It has been reported that hypouricemia may also predict the future progression and hence be an indicator of incipient nephropathy in Type 2 DM.^[15] Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein-Cholesterol (HDL-C) level that contributes to the development of atherosclerosis, which may be primary (genetic) or secondary and diagnosed by measuring plasma levels of total cholesterol (TC), TGs, and individual lipoproteins. It is traditionally classified by patterns of elevation in lipids and lipoproteins.^[16] Patients with type 2 DM are at greater risk of developing vascular diseases because of lipid changes. Aims of our study was to detect the correlation of serum uric acid level with glycaemic status as well as with lipid profile, and to find out various biochemical parameters, anthropometric measurements, blood pressure, measure the serum uric acid level and evaluate the association, if any, between the serum uric acid level and the factors measured of subjects.

Materials & Methods

A descriptive case –control study was conducted on the basis of inclusion and exclusion criteria, in department of Physiology, with the help of department of Medicine, Katihar Medical College,

Katihar, Bihar during period of June 2016 to July 2017.

A 100 diagnosed type 2 diabetes mellitus patients were enrolled in this study and 100 nondiabetes healthy individuals age and sex matched were taken as control. Male and female ratio was same. Entire subjects signed an informed consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought. Data was collected using random sampling.

Inclusion criteria of this study were subjects with age more than 40 years, diagnosed type II diabetes mellitus with no previous history of diabetic keto acidosis or pancreatitis. Exclusion criteria were patients suffering from Kidney disease, hepatic disorder, patients on diuretic therapy (mainly thiazides), history of alcoholism, suffering from myeloproliferative disorders, lymphoproliferative disorders and patients suffering from Psoriasis.

Methods

A detailed history was taken about their dietary pattern. Subjects were demonstrated steps of investigations properly. Next they were undergone investigations. All the reports of investigations and any altered status were explained to the patients.

History

A case data sheet was used to assess the clinical history, including past and present diseases both acute and chronic from all the subjects. The subjects were asked about their diet pattern. Emphasis was given on occupation, family history of diabetes mellitus, coronary artery diseases, musculoskeletal disorders, arthropathy. Their family income, history of addiction and level of physical activity was assessed.

Regarding present illness of the cases a meticulous history was taken about the chief complaints, symptoms of diabetes mellitus, duration of diabetes, symptoms of diabetes related complications e.g. decreased vision, pedal edema, chest pain, calf muscle pain, respiratory distress, increased frequency of micturation. Treatment

history, both for diabetes and its complications, in the form of drugs, dietary modification, lifestyle modification were also carefully noted.

Clinical assessment: It was started with general survey followed by systemic examinations.

General survey: Built, decubitus, anaemia, jaundice, cyanosis, clubbing, oedema, pulse, blood pressure, neck veins, neck glands, skin changes, height, weight.

Systemic examination: Emphasis was given upon the examination of cardiovascular system and endocrinal system. Other systems were also examined in brief.

Anthropometric measurements: Body mass index and waist- hip ratio.

Study tools was used in our study had stadiometer, measuring tape, weighing machine, Mercury sphygmomanometer.

Measurement of BMI: Height of subjects was measured with the help of a stadiometer. Weight was measured by the help of a weighing machine. BMI was calculated by dividing the weight of subjects in kilograms by the square of the height in meters. ^[17]

Measurement of waist-hip ratio: Waist circumference was measured (in centimetres) around the narrowest point between the lowest rib and hip when viewed from the front after exhaling. Hip circumference was measured (in centimetres) at the point where buttock is maximally extended, when viewed from the side. The ratio was calculated. ^[18]

Biochemical Investigations

Plasma Glucose was estimated by GOD-POD Method, End Point Assay and Kinetic Assay. ^[19]

Glycosylated Haemoglobin was estimated by ion exchanged resin method for quantitative determination of glycohaemoglobin in blood. ^[20]

Serum uric acid was estimated by Uricase / PAP method. ^[21]

LDL Cholesterol was estimated by direct determination of LDL Cholesterol. ^[22]

HDL Cholesterol was found by direct enzymatic method. ^[23] Triglycerides estimation was done by GPO / PAP method. ^[24]

Investigations proper

Blood glucose: Subjects had blood drawn after an overnight fast for fasting blood sugar. Analysis of post prandial blood sugar was done 2 hours after having a meal.

Plasma glucose was estimated by GOD/POD method by using spectrophotometer in the department of physiology, Katihar Medical College and Hospital. The kit for estimation of glucose was supplied by Crest Biosystems.

Serum HbA1C estimation: Serum glycosylated haemoglobin concentration of the patients was measured in the department of Biochemistry Katihar Medical College and Hospital. It was measured by ion –exchange HPLC with a glycosylated haemoglobin analysing system (DIAMAT, Bio-Rad Laboratories, Hercules,CA, USA).

Serum uric acid: Venous blood samples were taken in the morning with the subjects fasting for 12 hours. The uric acid was measured by the uricase method. ^[25]

Serum lipid profile: Serum lipid profile was measured of each subject in the department of Biochemistry by using different kits and spectrophotometer. Total cholesterol was estimated by CHOD-POD (cholesterol oxidase peroxidase) method. ^[26] A kit manufactured by LOGOTECH INDIA Pvt. Ltd was used.

Statistical Analysis

Data was analyzed by using SPSS software (Version 17). Mean \pm SD was observed. One way analysis of variance (ANOVA) with post hoc analysis using Tukey's multiple comparison test was used for parametric data. Pearson correlation coefficient (r) was used for correlation of data. P value was taken ≤ 0.05 for significant differences.

Observations & Results

A comparative cross sectional study was conducted on randomly selected 100((50: females and 50: males) diagnosed type2 diabetes mellitus patients. 100 subjects age and sex matched nondiabetic persons were taken as controls.

Table.1. Basic characteristics of the study subjects.

Parameters	Cases (N=100) MEAN \pm 2SD	Controls (N=100) MEAN \pm 2SD	P-value
Age(years)	56.70 \pm 14.10	56 \pm 13.24	0.869
BMI(kg/m ²)	27.35 \pm 5.15	26.49 \pm 4.25	0.011
WHR	0.92 \pm 0.22	1.77 \pm 0.12	0.336
SBP	135.96 \pm 32	127 \pm 28.4	<0.05*
DBP	83.72 \pm 24	78.6 \pm 21.54	0.002*

Intergroup comparison shown that samples was age matched (P>0.05). There was no significant difference of BMI, WHR among the groups. There was significant difference (i.e.P<0.05) of systolic and diastolic blood pressure among the case and control groups.

Table.2. Distribution of subjects according to age.

Age distribution(Years)	Cases Percentage (%)	Control Percentage (%)
<50	52.6 (n=30)	47.4 (n=27)
51-60	47.5 (n=38)	52.5 (n=42)
>60	50.8 (n=32)	49.2 (n=31)

The above table shows that among the age distribution <50, cases were 52.6%, controls 47.4%. In the age group 51-60, cases were 47.5% and controls 52.5%. Among the age group >60, cases were 50.8% ,controls 49.2%.

Table.3. Distribution of BMI among subjects.

BMI distribution	Case Percentage (%)	Control Percentage (%)
18.5-22.9	83.3 (n=10)	16.7 (n=2)
23-29.9	42.7 (n=64)	57.3 (n=86)
≥ 30	68.4 (n=26)	31.6 (n=12)

This table shows that in the obese BMI group (23-29.9) cases were 42.7% and controls 57.3%. Among the morbid obesity group (BMI>30) cases were 68.4% and controls 31.6%.

Table 4 Distribution of WHR among cases and controls.

WHR	Cases(%)	Controls(%)
>1	80.95(n=17)	19.04(n=4)
<1	46.37(n=83)	53.63(n=96)

Patients who had waist-hip ratio >1, cases were 80.95% and controls 19.04%. Of those who had waist –hip ratio <1, cases was 46.37% and controls 53.63%.

Table 5: Intergroup comparison of serum glucose levels between cases and controls.

Parameters	Cases MEAN±2SD	Controls MEAN±2SD	P value
FBS	210.51±106.14	75.62±20.66	<0.05*
PPBS	282.24±119.89	123.57±20.23	<0.05*
HbA1c	11.54±4.8	4.95±11.64	<0.05*

Above table shows significant differences (P<0.05) of fasting, post-prandial blood glucose levels and glycated haemoglobin level between cases and controls. All the values was significantly higher in cases (type2 diabetics) than control (normoglycaemics) group.

Table.6. Intergroup comparison of lipid profiles between cases and controls.

Parameters	Cases MEAN±2SD	Controls MEAN±2SD	P values
TOTAL CHOLESTEROL	264.43±89.52	176.13±52.04	<0.05*
LDL-C	180.28±89.78	106.20±51.78	<0.05*
HDL-C	39.70±23.08	56.71±16.14	<0.05*
TRIGLYCERIDE	204.78±97.42	148.40±67.38	<0.05*

Above table shows significant difference exists between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (Diabetics) was significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases is significantly lower than control group.

Table.7. Distribution of dyslipidemia among subjects.

Lipid Profile Status	Cases (%)	Control (%)
Dyslipidemic	67.5 (n=85)	32.5 (n=41)
Non-dyslipidemic	20.3 (n=15)	79.7 (n=59)

Table shows that among the dyslipidaemics 67.5% were cases and 32.5% controls. Among non-dyslipidaemics 20.3% were cases, 79.7% controls. So it can be inferred that distribution of

dyslipidaemia was more in cases (diabetics) than controls (non-diabetics).

Table.8. Intergroup comparison of serum uric acid levels between cases and controls.

Parameter	Cases MEAN±2SD	Controls MEAN±2SD	P-Value
Serum uric acid	3.55±2.436	7.38±2.142	<0.05*

When compared the serum uric acid, p value was found to be ≤ 0.05. Significant difference of serum uric acid exists between cases (diabetics) and controls (nondiabetics). It also shows that serum uric acid level was significantly lower in diabetic group than normoglycaemics.

Table 9. Correlation of serum uric acid level with blood glucose parameters.

Blood glucose parameters	Correlation of serum uric acid	
	Pearson correlation coefficient(r)	Significance (2-tailed)
Glycated haemoglobin	-0.918**	P<0.05*
Fasting blood sugar	-0.829**	P<0.05*
Post prandial blood sugar	-0.879**	P<0.05*

The above table shows a significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels. This correlations was also demonstrated by scatter diagrams.

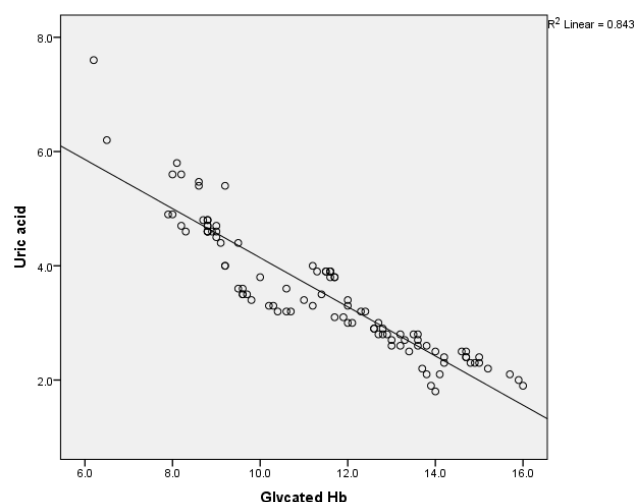


Figure.1. Correlation of uric acid with glycated Hb.

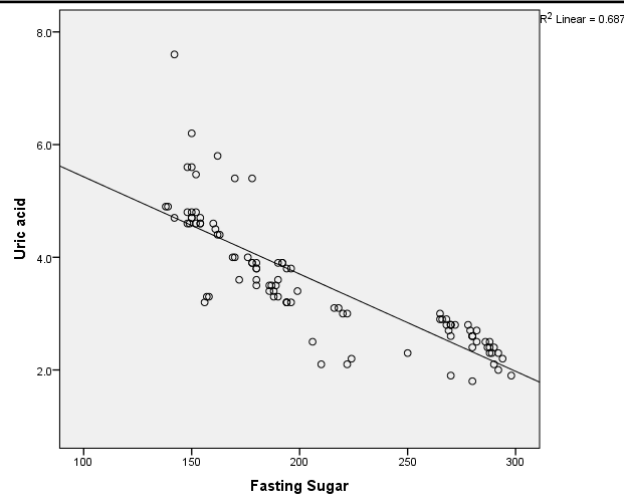


Figure.2. Correlation between uric acid and fasting sugar.

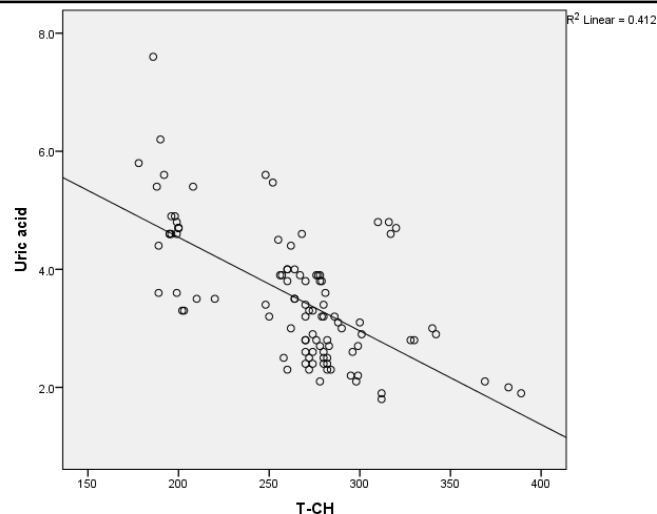


Figure.4 Correlation between uric acid and T-CH.

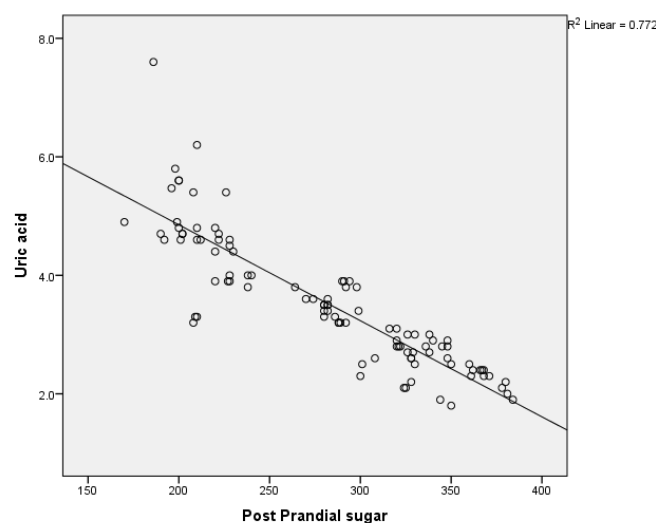


Figure.3. Correlation between uric acid and post prandial sugar

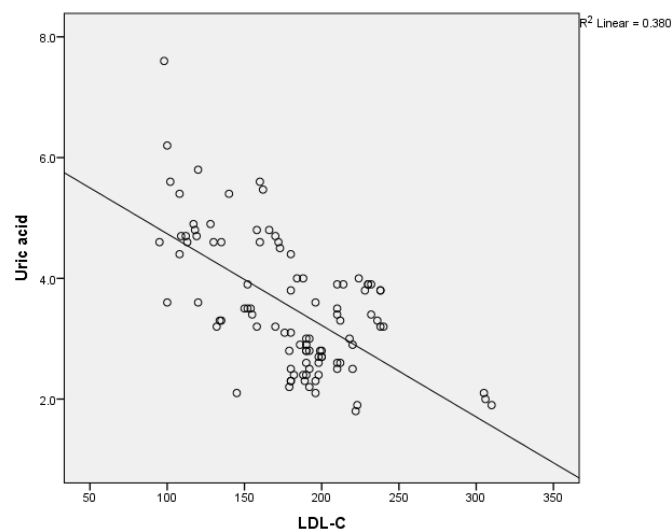


Figure.5. Correlation between serum uric acid and LDL-C.

Table.10. Correlation of serum uric acid level with serum lipid profile.

LIPID PROFILE	Correlation of serum uric acid	
	Pearson correlation coefficient(r)	Significance (2 – tailed)
Total cholesterol	-0.642**	0.000 *
LDL-cholesterol	-0.616**	P<0.05 *
HDL-cholesterol	0.651**	P< 0.05 *
Triglyceride	-0.721**	P<0.05 *

Table6. shows a significant strong negative correlation was exist between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was exist between serum uric acid level and HDL-cholesterol level. This correlations was also demonstrated by scattered diagrams.

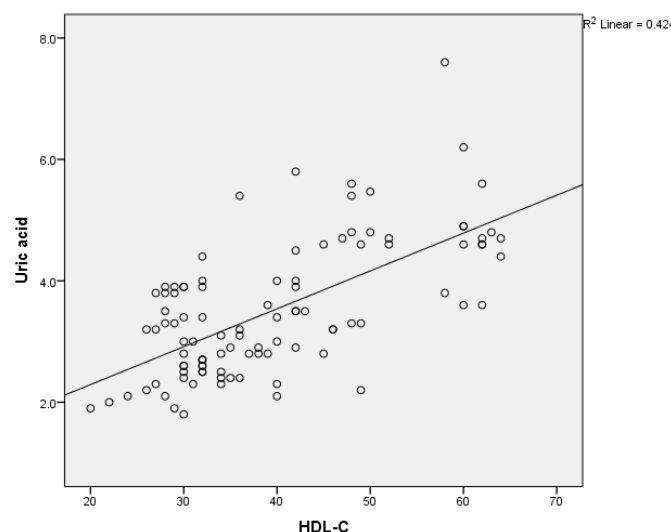


Figure.6. Correlation between uric acid and HDL-C.

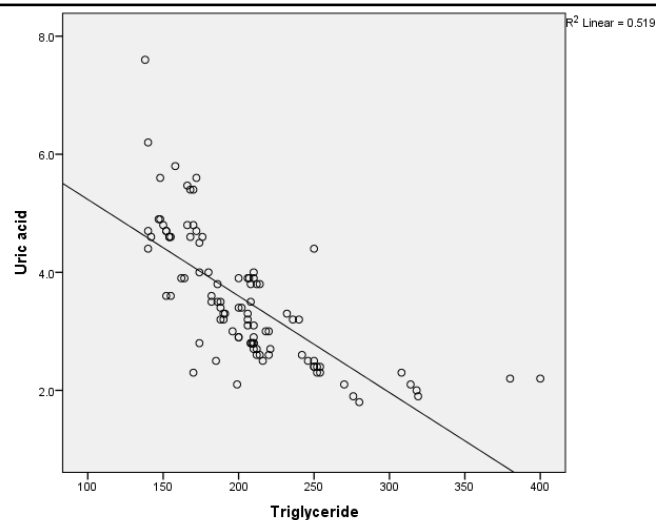


Figure.7. Correlation between uric acid and triglyceride.

Discussion

India, a developing Asian country with fast industrialization and a modern lifestyle is facing a grave problem in having the largest number of people with diabetes ^[2,3] which is estimated to reach 80 million by the year 2030. ^[4,5] It is close to becoming the diabetic capital of the world.

The incidence of cardiovascular disease is increased in individuals with type2 diabetes mellitus. The Framingham Heart study revealed a marked increase in coronary artery disease, myocardial infarction and sudden cardiac death in diabetes mellitus patients. The absence of chest pain (silent ischaemia) is also very common in patients of diabetes mellitus. So, to avoid such catastrophies various biochemical blood parameters, cardiological investigations should be done as a part of routine follow up.

Plasma uric acid, an end product of purine metabolism, is related to the purine bases of the nucleic acids. Its levels are genetically determined, but are influenced by multiple environmental factors. Previously it had been thought to be a metabolically inert end product without any physiological significance.

Recently, it has been shown that there is a definite relationship between hyperglycemia and uric acid levels. Studies done so far have shown that, in the early stages of diabetes, the levels were high and as the diabetic status progresses, there is a gradual

decline of uric acid levels in many patients. Studies showed that uric acid can act as an important water soluble antioxidant ^[27,28]. Urate, the soluble form of uric acid, can scavenge the superoxide and the hydroxyl radical and it also can chelate the transition metals. ^[29] There are evidences to suggest that low serum uric acid levels may precede the onset of diabetic retinopathy. It has been reported that hypouricemia may also predict the future progression and hence be an indicator of incipient nephropathy in Type 2 diabetes mellitus patients. In diabetes mellitus the uric acid excretion is increased due to osmotic diuresis caused by high plasma glucose level. As a result the plasma uric acid level is also decreased. However the exact relationship between the uric acid level and blood glucose parameters are still unknown. To further investigate these observations, we have conducted a case – control study on 100 type2 diabetes mellitus patients and 100 normoglycaemic healthy subjects to assess the blood glucose parameters, serum uric acid levels, lipid profiles and some anthropometric parameters.

In this study, we were found that there was no significant difference of Age, BMI, WHR among the groups. There was significant difference (i.e. $P < 0.05$) of systolic and diastolic blood pressure among the case and control groups. It was found that the cases had a mean age of 56.70 whereas the mean age among the controls was 56.54. It also shown that all the cases and controls were age matched ($P > 0.05$).

Among the age distribution < 50 , cases was 52.6%, controls 47.4%. In the age group 51-60, Cases were 47.5% and controls 52.5%. Among the age group > 60 , cases were 50.8%, controls 49.2%.

Regarding BMI it was suggest that in the obese BMI group (23-29.9) cases were 42.7% and controls 57.3%. Among the morbid obesity group ($BMI > 30$) cases were 68.4% and controls 31.6%. Mean \pm S.D. BMI of the cases (i.e. in Type2 diabetics) was 27.35 ± 5.15 and among the normoglycaemic control groups it was found to be 26.49 ± 4.25 .

In this study, who had waist-hip ratio >1 , cases were 80.95% and controls 19.04%. Of those who had waist-hip ratio <1 , cases were 46.37% and controls 53.63%. It was found that mean \pm S.D, waist-hip ratio of the cases were 0.92 ± 0.22 and among the control group it was 1.77 ± 0.12 .

Intergroup comparison of various blood glucose parameters between cases and controls: It was seen that fasting, post prandial blood sugar and glycated haemoglobin levels in cases were 210.51 ± 106.14 , 282 ± 119.89 and 11.54 ± 4.8 , and in controls were 75.62 ± 20.66 , 123.57 ± 20.23 and 4.95 ± 1.64 respectively. It was seen that all the blood glucose parameters were significantly higher in cases than control groups ($p < 0.05$). All these data confirms the selection of diabetics as cases.

Inter comparison of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels between cases and controls: It was shown significant difference exists between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (Diabetics) was significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases was significantly lower than control group ($p < 0.05$).

Distribution of dyslipidaemia among cases and controls: It was shown that among the dyslipidaemics 67.5% and 32.5% cases were in controls. Among non-dyslipidaemics 20.3% and 79.7% cases were in controls. So it could be inferred that distribution of dyslipidaemia was more in cases (diabetics) than controls (non-diabetics). There were various studies shown that high blood glucose level is associated with dyslipidaemia. One of the recent studies published in Indian Journal of Clin Biochem done by Mullugeta Y, Chawla R, Kebede T, in the year 2012 on 165 type2 diabetics were classified as good glycaemic control (group1) and poor glycaemic control (group2) on the basis of their blood HbA1C values.^[30] The group2 was characterized with serum triglyceride ($190.46 \pm$

15.20mg/dl), total cholesterol ($175.3 \pm 6.31 \text{mg/dl}$), as well as high LDL-cholesterol levels ($109.0 \pm 5.88 \text{mg/dl}$). Significant correlations was exists between HbA1c and dyslipidaemia, particularly serum TG ($r = 0.28, p < 0.05$), and between HbA1C and total cholesterol ($r = 0.310, p < 0.05$). So it can be said that our finding corroborates with the previous study results.

Intergroup comparison of serum uric acid level between cases and control groups: It was found that significant difference of serum uric acid level exists between cases (diabetics) and controls (nondiabetics). It was also shown that serum uric acid level was significantly lower in diabetic group than normoglycaemics. The mean and standard deviation of serum uric acid level in cases was 3.55 ± 2.436 and $7.38 (\pm 2.142)$. The p value was < 0.05 .

In previous studies, Godfredsen et al^[31] showed that diabetics had a 42% increase in renal uric acid excretion rate compared with normal. Diabetics had significantly lower mean serum uric acid concentrations. 17% of the diabetic patients had serum concentrations below the normal mean ± 2 standard deviations.

In a study in Israel by Herman and goldbourt in the year 1982 showed that prediabetic subjects had higher uric acid levels than non diabetics and that overt (clinically diagnosed) diabetics had lower uric acid levels than non diabetics.^[25]

Their finding of a negative association at the highest extreme of the glucose distribution was further supported by another study done by Derek G.Cook, A.G.Shaper, D.S.Thelle and T.P. Whitehead on 7735 British men aged 40-59 in British regional heart centre.^[26] The findings of our present study also agree with the findings of the previous studies.

In our study significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels ($p < 0.05$). The pearson's correlation coefficient (r) were $-0.918, -0.829, -0.879$ respectively.

In a study by Eiji Oda, Ryu Kawai et al on 2449 Japanese men and 1448 Japanese women the prevalence of metabolic syndrome and diabetes was calculated by the quartiles of serum level of uric acid levels. The results showed that prevalence of diabetes in third quartile was significantly lower than that in first quartile (lowest quartile) and the prevalence of diabetes in fourth quartile was significantly lower than that in first and second quartile in men. The prevalence of diabetes was not significantly different among the quartiles of uric acid in women. They concluded that serum uric acid level is negatively associated with diabetes in Japanese men.^[32]

In a study conducted by Qingdao Diabetes epidemiology study group, Qingdao, China over a total of 1288 men and 2344 women showed that serum uric acid levels declined with increasing fasting plasma glucose levels in individuals with diabetes mellitus, with standardized coefficient of -0.26 in men and -0.20 in women.^[33]

A recent study published in international journal of endocrinology in the year 2011, done by Pavani Bandaru and Anoop Shankar, showed the association between serum uric acid levels and diabetes mellitus in participants from the NHANES (n=18,825, 52.5% women). Serum uric acid was divided into quartiles. In multivariate logistic regression models, they found that higher serum acid levels were inversely associated with diabetes mellitus after adjusting for age, sex, race, smoking, alcohol intake, body mass intake, hypertension and serum cholesterol level. Compared to quartile 1 of serum uric acid, the odd's ratio (95% confidence interval) of diabetes mellitus was 0.48 (0.35-0.66; P trend < 0.0001). They concluded that higher serum uric acid levels were inversely associated with diabetes mellitus in a representative sample of US adults.^[34] Our findings agree with the results of the previous studies.

In our study, the correlation of serum uric acid levels with the total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels were performed. It shown a significant strong negative

correlation exists between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was existed between serum uric acid level and HDL-cholesterol level. The Pearson's correlation coefficients were -0.642 , -0.616 , 0.651 , -0.721 respectively.

In a study by J. Fang, M.H. Alderman on serum uric acid and cardiovascular mortality it shown that serum uric acid level had a continuous, independent, specific and significant negative relationship with cardiovascular mortality.^[13] Serum uric acid level was negatively correlated with serum total cholesterol, LDL-cholesterol and triglyceride level, and positively correlated with serum HDL-cholesterol level. Finding of our study results was similar with the findings of previous study.

In hyperglycemic state, the increasing glucose reabsorption may impair the tubular reabsorption of uric acid, as both glucose as well as filtered uric acid are reabsorbed at the same site, the proximal convoluted tubule. Continued hyperexcretion of uric acid due to hyperglycemia could deplete the uric acid pool and gradually reduce serum uric acid levels. Geoffrey Boner and Rieselbach^[35,36] concluded that the presence of glucose in the renal tubule lumen at a site distal to that of normal glucose reabsorption inhibits the tubular reabsorption of uric acid. Later in 1987, Schichiri et al emphasized that there is a possible mechanism of glomerular hyperfiltration, which brought about the increased renal clearance of urate and ultimately results in low serum uric acid level.

Limitation

- 1) The study population was small.
- 2) This was a cross sectional study with no follow up.
- 3) Study period was only one year.
- 4) The other parameters of nephrological and cardiological complications should be assessed (i.e. eGFR, ECG changes e.t.c.). That was not done in our study.

Summary & Conclusion

We were conducted a case –control study on 100 type 2 diabetes mellitus patients (taken as cases) with 100 non diabetic subjects(taken as controls) to assess whether there are any correlation between the serum uric acid level and the blood glucose parameters and the lipid profile, the serum uric acid level between the cases and controls are also compared. After inclusion of cases and controls, the whole procedure was explained to every subject and consent forms were duly signed. After that they underwent history taking, proper clinical examination, and special investigations (serum uric acid, blood glucose parameters, serum lipid profile). Then the data were collected and epilated on SPSS (Version-17) software and statistical analysis were done. Following results were obtained:

- Among the age distribution <50 years, 52.6% cases were in case group, and 47.4% in control group. In the age group 51-60 years, cases were 47.5% and controls were 52.5%. Among the age group>60 years, cases were 50.8%, controls were 49.2%.
- Regarding BMI, it was suggested that in the obese BMI group (23-29.9) case were 42.7% and controls were 57.3%. Among the morbid obesity group (BMI>30) cases were 68.4% and controls were 31.6%. BMI of the cases (i.e. in type2 diabetics) was 27.35 ± 5.15 and among the normoglycaemic control groups was 26.49 ± 4.25 .
- Those who have waist-hip ratio >1, cases was 80.95% and controls was 19.04%.Of those who had waist –hip ratio <1, cases was 46.37% and controls was 53.63%. Mean \pm S.D) waist-hip ratio of the cases was 0.92 ± 0.22 and among the control group it was 1.77 ± 0.12 .
- The inter group comparison of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels between cases and controls shown significant

difference between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (diabetics) were significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases was significantly lower than control group ($p < 0.05$).It also shown that among the dyslipidaemics 67.5% subjects was cases and 32.5% subjects were controls. Among non-dyslipidaemics 20.3% were cases and 79.7% were controls. So it can be inferred that distribution of dyslipidaemia was more in cases (diabetics) than controls (non-diabetics).

- A significant difference of serum uric acid level was exists between cases (diabetics) and controls (nondiabetics). It shown that serum uric acid level was significantly lower in diabetic group than in normoglycaemics. The mean \pm S.D. Serum uric acid level in cases was 3.55 ± 2.436 and in cases was 7.38 ± 2.142 .The p value was <0.05.
- There was a significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels ($p < 0.05$). The pearson's correlation coefficient (r) were -0.918,-0.829,-0.879 respectively.
- A significant strong negative correlation was existed between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was existed between serum uric acid level and HDL-cholesterol level. The pearson's correlation coefficients were -0.642,-0.616,0.651,-0.721 respectively.

Conclusion

Our study concluded that type 2 diabetes mellitus patients is a strong negative correlation between

blood glucose level and serum uric acid level. So it can be said that as the blood glucose level increases the serum uric acid level decreases. As the serum uric acid is an important water soluble antioxidant, low serum uric acid may give rise to much further oxidative damage to mainly small and large blood vessels. Serum uric acid level decreases the lipid profile worsens, which can give rise to cardiovascular complications in future. So, that serum uric acid level can be used as an important parameter to assess future cardiovascular risk in a type2 diabetes mellitus patient.

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