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Correlation Analysis of Serum Uric Acid with Insulin Resistance in Patients with Type-2 Diabetes

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Abstract

Objective: To evaluate correlations between SUA and insulin resistance in type-2 diabetes (T2DM) patients.

Methods: A total of 208 T2DM who were admitted between January 2015 and December 2017 to the Department of Endocrinology, Nanjing University of Chinese Medicine were analyzed herein. The selected parameters, including age, BMI, SUA, total cholesterol, TG, LDL-C, FBG, PBG, HbA1c, FINS and HOMA-IR, were estimated. Patients were categorized into a compliance group (HbA1c <7%) and a non-compliance group, (HbA1c ≥7%). For the HOMA-IR and SUA levels, Q1-Q4 was used to compare the indicators in each group. Furthermore, correlation and linear regression analysis were performed.

Results: Patients who had abnormal BMI and were obese presented with significant levels (p<0.05) of the selected biochemical parameters. Non-compliant patients had significantly higher (P<0.05) SUA, FBG and PBG levels, when relative to the compliance group. For HOMA-IR groups, from the Q1 to Q4 group, the BMI, FBG, TG and SUA levels gradually increased (P<0.05). For the SUA groups, from the Q1 to Q4 group, the BMI, FBG, PBG, TG and FINS levels increased, while age gradually decreased (P<0.05). In a correlation analysis, the SUA levels, and FBG, HbA1c, PBG, TGs, FINS, BMI and HOMA-IR had a positive correlation (P<0.05). The regression analysis showed that FBG, PBG, TGs, FINS, BMI, HbA1c and HOMA-IR are correlated to SUA levels (P<0.05).

Conclusion: The increase in SUA in T2DM is linked with the increase in insulin resistance caused by obesity, and abnormal glucose and lipid metabolism.

Keywords: Type-2 diabetes mellitus; Serum uric acid; Insulin resistance; Body mass index

1. Introduction

Diabetes mellitus is a multifactorial chronic disorder that causes other associated disorders, such as renal and cardiovascular complications, along with various types of microangiopathies, including other metabolic syndromes. It has been estimated by the International Federation of Diabetes that by 2040, the number of diabetes cases in adults would increase from 415 million to 642 million [1]. Furthermore, it has been understood from several cases that insulin resistance (IR) is an important cause of type-2 diabetes mellitus [2]. This is characterized by the sensitivity of the patient to reduce the insulin levels, which could be the state of the relatively low production of insulin [3]. With IR as a metabolic syndrome [4], the clinical condition of hyperuricemia is often accompanied by type-2 diabetes mellitus [5]. However, the negative effects of hyperuricemia have been gradually emerging in IR patients. This IR condition in patients results in other complications, such as gout attacks, which eventually lead to gout kidneys [6], thereby seriously affecting the patient's quality of life [7]. Several studies have revealed that people suffering from diabetes are not only prone to elevated blood uric acid levels, but also present with other various metabolic abnormalities [8].

Several researchers have discovered that serum uric acid (SUA) is a considerable biomarker of glycometabolic dysregulation, as these levels are correlated with glucose metabolism. However, sometimes, no linear association is found with SUA and blood glucose, as revealed by some varied

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experiments, instead being more akin to a bell curve-shaped relationship. That is, if the uric acid levels are elevated, there would be an increase in blood glucose level concentration in normal and prediabetic populations. However, in those with T2DM, SUA levels typically decrease with the increase in blood glucose concentration [9]. The basis for this inverse relationship in these T2DM patients, however, remains to be clarified. Since insulin levels are associated with uric acid levels, it has been observed in studies that the increase in SUA levels would elevate the rate of serum insulin levels in diabetic patients [10]. In another study, serum creatinine levels were shown to be higher in T2DM patients with elevated SUA levels. Approximately two-thirds of subjects with T2DM with elevated SUA levels had microalbuminuria. Furthermore, SUA and HbA1c levels were positively correlated. In addition, the study population had elevated uric acid levels with microalbuminuria in T2DM. Hence, it would be sensible to check the uric acid and urine albumin levels of subjects with T2DM, to prevent renal complications [11].

Although studies have provided a clue that insulin, uric acid and blood glucose are linked, it remains unclear whether insulin governs the association between SUA and blood glucose. Since there is no explanation for this mechanism, to date, individual biochemical parameters are examined for IR. Hence, the present study proposes a novel approach for the cumulative initiation and examination of biochemical parameters with SUA, in order to explore the association with IR in type-2 diabetic mellitus patients.

2. Patients and Methods

2.1 Study design

This study initially enrolled 1,123 T2DM patients who were hospitalized in the Department of Endocrinology, Affiliated Hospital of Integrated Traditional Chinese and Western Medicine, Nanjing University of Chinese Medicine. A signed written informed consent was provided by all patients before enrolment into the present study. The hospital ethics committee (2018LWKY062) approved this study, which was carried out from January 2015 to December 2017.

All patients who met the 1999 World Health Organization (WHO) diagnostic criteria of T2DM were included in the present study[12]. A specific exclusion criteria was designed, and patients were excluded if they suffered from acute and chronic complications of diabetes, with further serious complications, such as severe diabetic microangiopathy, diabetic macroangiopathy, diabetic neuropathy, and so on. Focus was given on patients who suffered from organ damage, such as liver and kidney dysfunction, cardiac insufficiency, and infections or other autoimmune diseases. Patients with a history of hypertension, patients who received diuretics, and patients whose blood pressure control does not reach the standard (more than 140/90 mmHg, 1 mmHg = 0.133 kPa) were excluded. Furthermore, patients who received diuretics medication that may affect the uric acid metabolism, and increase the risk of developing urine crystals, which would result in the onset of gout disorder, were also excluded. The patient is participating in sports rehabilitation or physical exercise of a certain intensity. Finally, 208 patients were included in the present study, which comprised of 113 male and 95 female patients. The duration of the disease of the patients was considered to range within 1-10 years, and the age of these patients ranged within 26-85 years old, with an average age of 58.42 ± 10.23 years old.

2.2 Methods

2.2.1 Comparison of general and biochemical parameters in the different body mass index (BMI) groups

We measured the heights and weights of recruited patients were measured to calculate the BMI as follows: BMI = weight (kg) / height^2 (m^2). Patients were categorized into three BMI groups: normal BMI, BMI <25 kg/m^2; overweight, BMI within 25-28 kg/m^2; obese, BMI >28 kg/m^2. These patients were further analyzed for biochemical variable such as fasting blood glucose, postprandial blood glucose, HbA1c, cholesterol, LDL-C, SUA, FINS and homeostasis model assessment-insulin resistance (HOMA-IR) levels.

2.2.2 Analysis of other biochemical parameters and demographic variables for patients in compliance (HbA1c <7%) and non-compliance (HbA1c ≥7%) groups

HbA1c patients in both the compliance and non-compliance groups were instructed to fast for approximately 8-10 hours. Then, early in the morning on an empty stomach, between 6:00 hours to 7:00 hours, the venous blood was drawn to detect the SUA, total cholesterol, triglycerides, and low-density lipoprotein-C (LDL-C), fasting blood glucose, glycosylated hemoglobin (HbA1c), and fasting insulin (FINS). Then, the oral glucose tolerance test was additionally conducted. After two hours, the venous blood was drawn to detect the postprandial blood glucose. According to the test results, the HOMA-IR was calculated as follows:

HOMA-IR fasting blood glucose (mmol/L) × FINS (mIU/L) / 22.5. Furthermore, the enzymatic method was used for the biochemical index, the glucose oxidase method was used for blood glucose, and ion exchange high pressure liquid chromatography was used for HbA1c.

2.2.3 Estimation of biochemical levels, age and BMI association in the HOMA-IR groups

According to the HOMA-IR level, the quartile method was used. This was divided into four groups according to three cut-off values (1.85, 3.01 and 4.83): Q1 group (0.43-1.85), Q2 group (1.85-3.01), Q3 group (3.01-4.83), and Q4 group (4.83-54.13). Based on the SUA level, and using the quartile method according to the three cut-off values (248, 293 and 347 μmol/L), this was divided into four groups: Q1 group (123-248 μmol/L), Q2 group (248-293 μmol/L), Q3 group (293-347 μmol/L), and Q4 group (347-631 μmol/L). Then, these were compared with other selected biochemical parameters, including age, BMI, fasting blood glucose, triglycerides, SUA, postprandial blood glucose, HbA1c, cholesterol, LDL-C and FINS.

2.2.4 Assessment of the different biochemical parameters and demographic variables with the SUA groups

According to the SUA levels, the quartile method for the four groups was used, from Q1 to Q4 groups, according to the cut off values, and the BMI, and other biochemical parameters, such as fasting blood glucose, postprandial blood glucose, triglyceride, FINS HbA1c, cholesterol, LDL-C and HOMA-IR, were calculated.

2.3 Statistical processing

Obtained data was assessed using the SPSS19.0 statistical software (IBM, Chicago, USA). Normally distributed data were given as $\overline{X} \pm S$, and were compared through t-tests and one-way ANOVAs as appropriate. Pairwise comparison was performed using the SNK method. Non-normally distributed data were given in median M (P25, P75). Comparisons of two groups were performed using the Kolmogorov-Smirnov test. Rank sum test was used when comparing multiple groups. The correlation analysis was performed using Person analysis. Linear regression was performed using the stepwise regression method. P<0.05 was the significance threshold.

3. Results

3.1 Comparison of general and biochemical parameters in the different BMI groups

Patients in the normal BMI group were compared with the abnormal and obese BMI groups, and it found that the fasting blood glucose (FBG), postprandial blood glucose, HbA1c, cholesterol, LDL-C, SUA, FINS, and HOMA-IR levels were elevated (P<0.05). It suggested that the blood glucose, blood lipids, and uric acid of obese individuals were higher than those of individuals with a normal BMI, with more evident insulin resistance in the obesity group. However, there was no statistical difference observed in patient's age and triglyceride level between groups (P>0.05, Table 1).

3.2 Analysis of other biochemical parameters and demographic variables in the compliance (HbA1c <7%) and non-compliance (HbA1c >7%) groups

In the present study, based on the HbA1c % in the patient's blood sample, patients were assigned to the compliance group, when their HbA1c was below 7%, and the non-compliance group, when their HbA1c was above 7%. These two groups were examined for other biochemical parameters, and it was found that the FBG, postprandial blood glucose and SUA levels of patients were significantly wlwcarws in the non-compliance group, when compared to the compliance group (P<0.05), which meant that the higher the glycation of hemoglobin, the less well-controlled the patient's glucose and uric acid. However, there was no statistical significant difference in terms of age, BMI, cholesterol, triglyceride, LDL-C, FINS and HOMA-IR (P>0.05, Table 2). Blood glucose differences had little effect on these related indicators of the two groups.

3.3 Estimation of the biochemical levels, age and BMI in association with the HOMA-IR groups

Patients in the Q1-Q4 groups were examined by quartile analysis. It was found that the association with the BMI, fasting blood glucose, triglycerides and SUA levels gradually increased (P<0.05). This showed that the more obvious insulin resistance was, the more likely patients were to develop obesity, hyperglycemia, hyperuricemia and hypertriglyceridemia. However, there was no significant difference, in terms of age, postprandial blood glucose, HbA1c, cholesterol, LDL-C and FINS (P>0.05, Table 3), indicating that the difference in HOMA-IR had little effect on these related indicators of the two groups.

3.4 Assessing the different biochemical parameters and demographic variables with the

SUA groups

The Q1-Q4 groups of patients were assessed using quartile analysis for the selected biochemical and demographic variables. It was observed that the BMI, FBG, postprandial blood glucose, triglyceride and FINS levels increased, and that age gradually decreased with a statistical significance (P<0.05). This showed that the higher the blood uric acid level, the more likely the patient would be obesity, hyperglycemia, hypertriglyceridemia, and hyperinsulinemia. However, there was no difference in the HbA1c, cholesterol, LDL-C and HOMA-IR levels (P<0.05, Table 4). It indicated that the level of uric acid had little effect on glycosylated hemoglobin, cholesterol and HOMA-IR.

3.5 Correlation analysis

When these patients were analyzed, the SUA levels and FBG (r = 0.186, P=0.008), postprandial blood glucose (r = 0.234, P=0.009), HbA1c (r = 0.183, P=0.009), triglycerides (r = 0.449, P<0.001), FINS (r = 0.259, P<0.001), BMI (r = 0.239, P=0.001) and HOMA-IR (r = 0.161, P=0.022) were positively correlated, and there was significant correlation with the disease condition. However, there was no statistical correlation with age (r = -0.131, P=0.062), cholesterol (r = 0.023, P=0.741) and LDL-C (r = -0.020, P=0.781). This meant that diabetic patients have poor control of blood sugar and blood lipids, and obvious insulin resistance, the more likely they were to increase blood uric acid levels.

3.6 Linear regression analysis

After screening factors correlated to SUA by single-factor correlation analysis, it was found that FBG, postprandial blood glucose, triglycerides, FINS, BMI, HbA1c and HOMA-IR were correlated to the SUA levels (P<0.05). On the other hand, performing the linear regression analysis for the above data, it was found that BMI and HbA1c are risk factors that elevate the SUA levels in patients with T2DM (Table 5). That was, diabetic patients with high body weight and high glycosylated hemoglobin were more likely to have elevated blood uric acid.

4. Discussion

In our study, it was found that with a higher the BMI, there is a probability that the patient's glucose and lipid metabolism would be abnormal, and that there would be significant increase in HOMA-IR and SUA levels. The present study results are consistent with those of Mele *et al.* [13], in which it was understood that obesity drives an increase in SUA levels. The present study revealed that patients with abnormal HbA1c have significantly higher SUA levels, relative to patients in the normal group, and these uric acid levels were positively correlated with HbA1c. This suggests that for diabetic patients with better blood sugar control, the risk of developing hyperuricemia is lower [14]. The present results were similar to those of *Cui et al.* [15]. In this study, in the HOMA-IR groups, it was found that more IR was observed with higher BMI, fasting blood glucose, triglyceride and SUA levels.

The linear regression analysis suggests that HbA1c, BMI and triglycerides are risk factors for hyperuricemia in patients with T2DM. Therefore, diabetic patients should actively control weight, lower the blood sugar and blood lipids, and prevent the occurrence of hyperuricemia.

With the increase in SUA level, the BMI and HOMA-IR gradually increases, and FBG, postprandial blood glucose, triglycerides and FINS also significantly increases. Since uric acid has a direct correlation with these indicators.

The present study revealed that SUA levels in type-2 diabetes mellitus patients can be significantly elevated[66][17]. Since uric acid is a terminal purine metabolism byproduct, its accumulation can lead to the occurrence of various metabolic diseases, including T2DM [18]. IR is closely linked to the pathogenesis of T2DM [19]. Hence, we herein aimed to observe the relationship between SUA levels and IR in patients with T2DM.

The mechanism of the influence of IR on glucose and lipid metabolism includes the following: The sensitivity in the surrounding tissues decreases insulin production, which leads to the abnormal metabolism of glucose in the body. At the same time, liver glucose output is abnormal, resulting in increased blood sugar. Meanwhile, the body was accompanied by blood lipid disorders, leading to diabetes, and even the occurrence of metabolic syndrome [20][21].

Obesity leads to increased blood uric acid levels, mainly including three aspects. First, obese people have a relatively higher intake than normal people, and the purine synthesis in the body increases, resulting in increased uric acid synthesis [20]. Second, obese people are often accompanied by abnormal lipid metabolism, which is mostly hypertriglyceridemia, and the free fatty acids of triglyceride metabolites have a role of activating purine synthesis, thereby increasing the level of SUA [23][24].[23][24]. Third, insulin resistance is often present in obesity, and IR promotes the conversion of glycolysis intermediate products to ribose pyrophosphate and ribose 5-phosphate, increasing uric acid production [25]. Therefore, high

uric acid levels are closely correlated to body weight and IR.

The mechanism of IR that leads to hyperuricemia may be correlated to the following conditions. When the body is in the state of IR, this can promote the decomposition of fat. This would allow a large amount of free fatty acids to be released into the blood, and cause the synthesis of fatty acids to be stimulated, which ultimately leads to the increase in purine synthesis, and the promotion of the excessive production of uric acid [26][27]. On the other hand, the biological active substances released by adipose tissues include inflammatory factors, adiponectin, endogenin, leptin, etc. [28]. Hence, these factors would also promote the synthesis or reabsorption of uric acid [29]. IR leads to hyperinsulinemia [30]. This increases the reabsorption of uric acid through the kidneys, which in turn reduces uric acid excretion [31].

High uric acid can lead to IR, and this mechanism includes two aspects: [32][33]: (1) High uric acid causes a decrease in nitric oxide in the endothelium of blood vessels, leading to endothelial dysfunction [34], and in turn induces insulin resistance. (2) Uric acid can reduce the synthesis of adiponectin 0, leading to endocrine disorders in fat cells, inflammatory reactions [36] and oxidative stress [37], and ultimately causing IR. IR can lead to abnormal glucose and lipid metabolism [38]. At the same time, high uric acid reduces insulin secretion by inhibiting islet cell function, resulting in abnormal blood glucose [39]. However, the present study did not reveal that uric acid is correlated to cholesterol levels. Hence, our results differed from the results reported by *Fu et al.* [40]. This may be correlated to the use of lipid-lowering drugs by patients.

The limitations of this study were the single-center retrospective nature of the study, and the relatively limited number of included patients. The future prospect of the present study is to conduct in-depth research in the later stage of research, in which a number of cases and relevant observation indicators would be selected to improve future studies.

Conclusion

The increase in SUA in T2DM is associated with the increase in IR caused by obesity, and abnormal glucose and lipid metabolism. This study suggests that in our clinical work, in addition to paying attention to the patient's blood glucose, other metabolic indicators should also be considered comprehensively to avoid the early occurrence of long-term complications and comorbidities. However, further research must be carried out by considering the genetic status of the patient through selecting few associated genes; the gender bias involved the life style of the patient, and the socioeconomic status and associated clinical risk factors, which could enhance the significance of the study.

Abbreviations

BMI Body mass index
FBG Fasting blood glucose
FINS Fasting insulin
HOMA-IR Homeostasis model-assessment of insulin resistance
HbA1c Glycosylated hemoglobin
LDL-C Low density lipoprotein-cholesterol
PBG Postprandial blood glucose
SUA Serum uric acid
TG Triglycerides

Conflicts of interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

Wen Cao, Rendong Zheng and Ling Lv contributed equally to this work.

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Date availability

The date required to reproduce these findings cannot beshared at this time as the date also forms part of an ongoing study.

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Tables

Table 1. **Comparison of general indexes and biochemical indexes in the different body mass index groups ([$\bar{x} \pm s$]/M [P25, P75])**

Groups	n	Age (year)	Course of disease(year)	Fasting blood glucose (mmol/L)	Postprandial blood sugar (mmol/L)	HbA1c (%)	Cholesterol (mmol/L)	Triglyceride (mmol/L)	LDL-C (mmol/L)	SUA (μmol/L)	FINS (mIU/L)	HOMA-IR
Normal group	98	59.34±8.10	6（4，8）	7.93 (7.08,8.79)	14.00 (12.78,16.17)	8.70 (7.40,10.00)	4.44±0.77	1.39 (0.87,1.92)	2.85±0.76	282.59±70.44	6.09 (3.92,8.78)	2.24 (1.54,3.74
Abnormal group	67	59.09±10.62	5（3，8）	8.43a (6.90,9.95)	15.36 a (13.08,18.21)	9.90 a (9.10,10.90)	4.64±1.05 a	1.62 (1.05,2.80)	2.96±0.97 a	319.78±75.62 a	8.36 a (5.48,12.00)	3.14 a (2.01,4.84
Obese Group	43	55.33±13.31	5（4，7）	8.81 a (7.49,11.13)	17.31 a (14.60,19.99)	10.05 a (8.40,11.65)	4.88±1.02 a	1.45 (1.15,2.14)	3.27±0.85 a	320.55±86.28 a	11.73 a (8.98,18.04)	3.91 a (3.19,6.33
t/Z		2.739	0.911	0.911	8.867	3.497	0.874	21.860	1.487	8.98	36.668	25.323
P-value		0.082	0.634	0.041	<0.001	0.014	0.039	0.062	0.013	0.002	<0.001	<0.001

HbA1c: glycosylated hemoglobin, LDL-C: low density lipoprotein, SUA: Serum uric acid, FINS: fasting insulin, HOMA-IR: the steady-state model assessment-insulin resistance index, *vs.* normal group，[a]*P*<0.05. Table 1 shows that compared with the normal group, fasting blood glucose, postprandial blood glucose, HbA1c, cholesterol, LDL-C, SUA, FINS, HOMA-IR levels gradually increased in the abnormal group and obese groups.

Table 2. **Comparison of general and biochemical indicators in the different HbA1c groups ([$\bar{x} \pm s$]/M [P25, P75])**

Group	N	Age（year）	Course of disease (year)	BMI (kg/m^2)	Fasting blood glucose (mmol/L)	Postprandial blood sugar (mmol/L)	Cholesterol (mmol/L)	Triglyceride (mmol/L)	LDL-C (mmol/L)	SUA (μmol/L)	FINS (mIU/L)	HOMA-IR
Compliance group	29	56.17±8.05	6（3,9）	26.71 (23.32,28.70)	6.92 (5.71,7.99)	13.14 (10.93,15.53)	4.59±1.12	1.56 (0.90,2.42)	2.95±1.02	287 (245,342	10.38 (5.82,11.48)	2.61 (2.17,3.60)
Non-compliance group	179	58.79±10.55	6（4,8）	25.10 (22.92,27.29)	8.70[a] (7.49,10.62)	16.50[a] (14.03,19.11)	4.73±0.97	1.45 (1.05,2.09)	3.11±0.86	328[a] (269,384	7.78 (5.10,11.72)	3.02 (1.84,4.90)
t/Z		1.525	0.680	1.147	2.204	2.083	0.439	0.495	2.633	1.444	1.012	1.148
p-value		0.203	0.744	0.144	<0.001	<0.001	0.499	0.967	0.380	0.031	0.257	0.143

BMI: body mass index, LDL-C: low density lipoprotein, SUA: Serum uric acid, FINS: fasting insulin, HOMA-IR: the steady-state model assessment-insulin resistance index, *vs.* normal group，[a]*P*<0.05. Table 2 shows that the fasting blood glucose, postprandial blood glucose and SUA levels in the non-compliance group were significantly higher than those in the compliance group.

Table 3. **Comparison of general and biochemical indicators in the different HOMA-IR groups ([$\bar{x} \pm s$]/M [P25, P75])**

Group	n	Age（year）	Course of disease(year)	BMI (kg/m^2)	Fasting blood glucose (mmol/L)	Postprandial blood sugar (mmol/L)	HbA1c(%)	Cholesterol (mmol/L)	Triglyceride (mmol/L)	LDL-C (mmol/L)	SUA (μmol/L)	FINS (mIU/L)
Q1	52	58.73±8.27	6（4,8）	23.84(21.79,26.22)	7.51(6.49,9.47)	16.09(13.00,18.38)	9.40(8.40,10.50)	4.80±1.15	1.14(0.79,1.54)	3.23±0.89	273.65±57.95	3.78(2.65,4.92)
Q2	52	56.02±9.79	6（4,8）	24.89(22.62,22.94)	8.39(6.83,9.83)	16.10(13.96,18.87)	9.80(7.85,11.20)	4.87±0.94	1.58(1.20,2.07)	3.18±0.89	303.73±79.03	6.52(5.46,7.90)
Q3	52	59.90±10.83	5（3,7）	26.29(24.29,28.70)	8.64(7.51,10.63)	15.38(13.16,18.47)	9.55(7.95,10.80)	4.48±0.77	1.59(1.07,2.48)	2.85±0.78	306.22±70.82	9.84(8.05,11.15)
Q4	52	59.06±11.71	6（3,8）	26.55[a](24.28,29.39)	9.25 [a](8.20,11.50)	16.21(13.34,19.90)	10.30(7.90,11.50)	4.69±1.05	1.67 [a](1.04,2.74)	3.08±0.95	324.71±91.55[a]	16.51(12.76,28.80)
F/χ^2		1.545	1.122	29.625	30.398	2.940	0.979	0.348	17.134	1.724	2.867	182.479
p-value		0.243	0.772	<0.001	<0.001	0.833	0.727	0.216	0.001	0.126	0.009	<0.001

BMI: body mass index, HbA1c: glycosylated hemoglobin, LDL-C: low density lipoprotein, SUA: Serum uric acid, FINS: fasting insulin, *vs.* Q1 group，[a]*P*<0.05. Table 3 shows that from Q1 group to Q4 group, the body mass index, fasting blood glucose, triglycerides and serum uric acid levels gradually increased.

Table 4. **Comparison of general indexes and biochemical indexes in different blood uric acid groups [($\bar{x} \pm s$)/ M (P25, P75)]**

Group	N	Age year）	（Course of disease (year)	BMI (kg/m^2)	Fasting blood glucose (mmol/L)	Postprandial blood sugar (mmol/L)	HbA1c(%)	Cholesterol (mmol/L)	Triglyceride (mmol/L)	LDL-C (mmol/L)	FINS (mIU/L)	HOMA-IR
Q1	52	60.04± 9.93	5（4,8）	24.02 (22.49,29.66)	8.29 (6.90,9.64)	14.81 (12.04,17.37)	10.00 (8.77,11.80)	4.60 (4.23,5.38)	1.10 (0.79,1.48)	3.14± 0.81	5.51 (3.95,9.74)	2.25 (1.50,,3.95)
Q2	52	60.30± 8.97	6（4,8）	24.75 (22.35,28.22)	8.43 (6.81,9.69)	15.06 (13.25,18.09)	9.50 (8.03,11.17)	4.59 (3.88,5.18)	1.28 (0.82,1.84)	3.01± 0.95	8.00 (4.12,14.15)	2.99 (1.71,5.10)
Q3	52	58.65± 8.09	6（3,7）	25.91 (24.21,27.21)	8.43 (7.32,10.02)	16.25 (13.43,19.12)	9.50 (7.70,11.60)	4.89 (4.26,5.50)	1.62 (1.09,3.66)	3.14± 0.90	8.12 (5.89,12.40)	3.23 (2.04,4.91)
Q4	52	55.02± 13.03	6（4,8）	26.37[a] (24.49,28.41)	9.80[a] (7.79,11.37)	17.49[a] (14.46,19.99)	9.40 (7.60,11.50)	4.60 (4.05,5.35)	1.86[a] (1.45,2.55)	3.03± 0.91	9.36[a] (7.17,12.15)	3.29 (2.45,4.88)
F/χ^2		2.905	5.639	9.428	8.707	12.409	3.955	1.259	39.010	0.299	16.122	6.831
p-value		0.036	0.131	0.024	0.033	0.006	0.266	0.739	<0.001	0.826	0.001	0.077

BMI: body mass index, HbA1c: glycosylated hemoglobin, LDL-C: low density lipoprotein, FINS: fasting insulin, HOMA-IR: the steady-state model assessment-insulin resistance index, *vs.* Q1 group, [a]*P*<0.05. Table 4 shows that from Q1 group to Q4 group, the body mass index, fasting blood glucose, postprandial blood glucose, triglyceride and FINS levels increased, while age gradually decreased.

Table 5. **Linear regression analysis of elevated blood uric acid in patients with type 2 diabetes**

Variate	*B value*	β *value*	T *value*	*p-value*	95%CI	
					Lower limit	Upper limit
Constant	225.339	44.895	5.019	<0.001	136.805~313.874	
Triglyceride	11.010	3.446	3.195	0.002	4.213~17.806	
BMI	3.989	1.428	2.793	0.006	1.172~6.805	
HbA1c	4.617	1.990	2.320	0.021	0.693~8.542	

BMI: body mass index, HbA1c: glycosylated hemoglobin