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Original Research Article

Study of Serum Uric Acid and Adenosine Deaminase as Markers of Oxidative Stress in Patients of Type 2 Diabetes Mellitus

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ABSTRACT

Background: Diabetes Mellitus (DM) is a common metabolic disease due to absolute or relative insulin deficiency. Oxidative stress plays a major role in pathophysiology of type 2 DM and its prevalence is increased due to lifestyle and obesity. In past years different studies has been done to identify various oxidative stress markers. Aim of this study is to evaluate levels of serum Uric Acid (UA), Adenosine Deaminase (ADA), Fasting Plasma Glucose (FPG) and HbA1c in patients of Type 2 Diabetes Mellitus and healthy subject and also to find out role of UA and ADA as a marker of oxidative stress in type 2 DM.

Material and methods: We measured the serum level of Uric Acid, ADA, HbA1c and FPG in 100 patients with type 2 diabetes and 50 healthy controls. Subjects included in study group were divided on the basis of duration of diabetes mellitus. Group 1 included subjects with type 2 DM < 1 year and Group 2 included subjects with type 2 DM > 5 year.

Results: The difference in FPG, ADA and HbA1c were highly significant ($p<0.05$) in type 2 diabetic patients (< 1 year) in comparison to control group while the difference in uric acid is not significant in diabetes patients with < 1 year duration and healthy subjects. The difference in FPG, ADA, Uric Acid and HbA1c were highly significant ($p<0.05$) in type 2 diabetic patients (> 5-year duration) in comparison to control group. While comparing two diabetes groups difference in FPG, ADA, Uric Acid and HbA1c were also highly significant ($p<0.05$).

Conclusion: Uric acid as a predictor of oxidative stress become significant as the duration of diabetes increases while ADA is statistically significant even during early onset of diabetes mellitus.

Keywords: Type 2 Diabetes Mellitus, Oxidative stress, Uric Acid, Adenosine Deaminase

INTRODUCTION

Diabetes is not one disease, but rather is a heterogeneous group of syndromes characterized by an elevation of fasting blood glucose caused by a relative or absolute deficiency of insulin.[1] The burden of diabetes is high and increasing globally, and in India, it is due to increasing prevalence of overweight/obesity and unhealthy lifestyles. The estimates in 2019 showed that 77 million individuals had diabetes in India, which is expected to rise to over 134 million by 2045. Current global statistics shows

that 463 million and 374 million individuals have diabetes and impaired glucose tolerance (IGT), a prediabetic condition. These numbers are estimated to increase to 700 million people with diabetes and 548 million people with IGT by 2045, which presents a 51% increase compared to 2019.[2]

In humans, uric acid is the major end product of purine nucleosides adenosine and guanosine. Measurement of uric acid is predominantly used in diagnosis and monitoring of gouty arthritis and pregnancy- induced hypertension.[3] Hyperglycemia causes oxidative stress which is associated with the pathophysiology of DM. Hadeel Ahmed Shawki et al concluded that levels of oxidative stress markers such as uric acid and malondialdehyde (MDA) were higher and associated with increased risk of diabetes and diabetic retinopathy development.[4] However, as a member of metabolic syndrome (MetS), uric acid (UA) could worsen insulin resistance by disturbing insulin-stimulated glucose uptake.[5] And, two meta-analysis revealed that elevated Serum Uric Acid has been an independent risk factor for the development of type 2 diabetes (T2DM).[6,7]Adenosine deaminase (Adenosine Aminohydrolase, EC 3.5.4.4) is an enzyme of purine metabolism which acts on adenosine and other adenosine nucleoside analogues which catalyze its hydrolytic cleavage into inosine and ammonia, so it causes reduction in the levels of adenosine.

Adenosine mimics the action of insulin on glucose and lipid metabolism in adipose tissue and the myocardium, while it inhibits the effect of insulin on total hepatic glucose output, which suggests that adenosine, causes local insulin resistance in the liver.[8] Seppo Lehto et all in 1977 explained in their study that hyperuricemia is a strong predictor of stroke events in middle-aged patients with Non-Insulin Dependent Diabetes Mellitus independently of other cardiovascular risk factors.[9] H. K. Choi and E. S. Ford used data from 14 664 participants aged 20 years and older in The US Third National Health and Nutrition Examination Survey (1988–1994)and demonstrated that Serum uric acid levels increased linearly with increasing fasting serum C-peptide levels, serum insulin levels or insulin resistance (multivariate P-values for trend, <0.001).[10] Gitanjali et al., conducted study on known patients of type 2 DM attending civil hospital, Patiala and concluded that ADA, Free fatty acids (FFA), MDA were significantly higher and correlated with increased HbA1c levels.[11] Other Studies have also reported elevated ADA activity in type 2 diabetes which concluded ADA as a marker of oxidative stress and lipid peroxidation in diabetes.[8,12,] Hence present study was designed to evaluate both serum uric acid and ADA as a markers of oxidative stress in type 2 Diabetes mellitus patients.

MATERIAL AND METHODS

Study area: In the present cross-sectional study, we included 100 type 2 diabetes mellitus patients. They are grouped according to the duration of disease, Group 1(50 subject) consists of patients having diabetes for < 1 year and group 2(50 subject) consist of patients having diabetes for > 5 year.

Inclusion criteria: Patients in age group of 35 -74 years of either sex included. Patient with confirmed case of type 2 diabetes having duration less than 1 year and on oral hypoglycaemic drugs. Patient with confirmed case of type 2 diabetes having duration 5 or more years and on oral hypoglycaemic drugs. Patient who is able to give informed consent.

Exclusion criteria: Type 1 Diabetes Mellitus, Acute complications of type 2 diabetes or patients on insulin treatment, Renal Failure, Hypertension or any other Pre-existing heart disease, Smoking, Chronic use of medicine e.g., glucocorticoids, any systemic disease e.g., Gout, Rheumatoid Arthritis, Skeletal muscle injury and person who denied to give consent. A group of 50 normal, healthy individuals from the same population served as controls. The duration of study was 9 months. The study protocol was approved by an Institutional review board of Maharaja Krishna Kumarsinhji Bhavnagar University. After enrolling in the study, a detailed medical history and the informed consent were obtained. A thorough explanation of the procedure of this study was given to the subject.

Venous Blood samples were collected from forearm after minimum 8 hour fasting state in plain vacutte (3 ml) for estimation of Serum ADA and Serum uric acid, for FPG Sodium Flouride vacutte (2 ml) was used while whole blood collected in EDTA vacutte (2 ml)

for estimation of HbA1c. Fasting plasma glucose were analyzed by Glucose Oxidase- Peroxidase (GOD POD) method[13], estimation of serum ADA done by Guisti Colorimetric Method[14] , estimation of serum uric acid was done by uricase method[15] and HbA1c was estimated by Immunoturbidimetric latex method[16].

All samples were run on fully automated biochemistry analyser Ilab 650 at Clinical Biochemistry Section, Laboratory Services Sir Takhtsinhji Hospital, Bhavnagar, Gujarat.

Statistical analysis: Numerical variables are reported in terms of mean and standard deviation. Comparison between two groups was made with the unpaired student-t test. Statistical software SPSS 20.0 version was used and P value <0.05 was considered as statistically significant.

RESULTS

Table-1: Distribution of subjects (Gender wise)

Type 2 diabetes patients (< 1 year duration)		Type 2 diabetes patients (>5 year duration)		Controls	
Male	Female	Male	Female	Male	Female
24 (48%)	26 (52%)	28(56 %)	22(44 %)	23(46 %)	28(54 %)

Mean age group for type 2 diabetes patients (< 1 year duration) was 52.56± 9.2, for diabetes patients (>5-year duration) was 52.22±9.6 and for controls it was 51.56±8.9 (Table-2).

Table-2: Distribution of subjects (Age wise)

Type 2 diabetes patients (< 1 year duration)		Type 2 diabetes patients (>5-year duration)		Controls	
35-50 years	>50 years	35-50 years	>50 years	35-50 years	>50 years
22	28	25	25	23	27

The difference in FPG, ADA and HbA1c were highly significant ($p<0.05$) in type 2 diabetic patients (< 1 year) in comparison to control group while the difference in uric acid is not significant in diabetes patients with < 1 year duration in comparison to control group (Table-3).

Table-3: Comparison of UA, ADA, FPG and HbA1c in type 2 diabetes patients (< 1 year duration) and healthy subjects

Parameter	Biological Reference Interval	Type 2 diabetes patients (< 1 year duration)	Healthy subjects	Statistical Significance
Uric Acid	Male: 3.5-7.2mg/dl Female:2.6-6.0 mg/dl	4.64 ± 1.31	4.118 ± 1.26	t = 2.05 p > 0.05
ADA	0-15 U/L	20.23 ± 7.422	16.48 ± 6.30	t = 2.72 **p<0.05
FPG	70-100 mg/dl	124.66 ± 27.70	90.88 ± 6.48	t = 8.395 **p<0.05
HbA1c	4.0-6.0 %	6.13 ± 0.55	4.842 ± 0.57	t = 11.50 **p<0.05

Table-4: The difference in FPG, ADA, Uric Acid and HbA1c were highly significant (p<0.05) in type 2 diabetic patients (> 5-year duration) in comparison to control group.

Parameter	**Biological Reference Interval**	**Type 2 diabetes patients (>5-year duration)**	**healthy subjects**	**Statistical Significance**
Uric Acid	Male: 3.5-7.2mg/dl Female:2.6-6.0 mg/dl	5.38 ±1.56	4.118 ± 1.26	**t= 4.43** **p<0.05
ADA	0-15 U/L	24.52 ± 9.73	16.48 ± 6.30	**t = 4.91** **p<0.05
FPG	70-100 mg/dl	165.06 ± 90.60	90.88 ± 6.48	**t= 5.77** **p<0.05
HbA1c	4.0-6.0 %	9.15 ± 2.14	4.842 ± 0.57	**t= 13.74** **p<0.05

The difference in FPG, ADA, Uric Acid and HbA1c were highly significant (p<0.05) in type 2 diabetic patients (> 5-year duration) in comparison to control group (Table-4).

Table-5: Comparison of UA, ADA, FPG and HbA1c in two groups of type 2 diabetes patients

HTN and DM	**Body Mass Index**			**Total**	**p value**
	Underweight	**Normal**	**Obese**		
Blood pressure					
Normal	14 (66.6%)	70 (86.41%)	39 (67.2%)	123 (76.8%)	0.014*
Hypertension	7 (33.3%)	11 (13.5%)	19 (32.7%)	37 (23.12%)	
Total	21 (100%)	81 (100%)	58 (100%)	160 (100%)	
Diabetes mellitus					
Normal	16 (76.2%)	72 (88.8%)	45 (77.5%)	133 (83.1%)	0.14
Diabetes mellitus	5 (23.8%)	9 (11.2%)	13 (22.4%)	27 (16.9%)	
Total	21 (100%)	81 (100%)	58 (100%)	160 (100%)	

While comparing two diabetes groups difference in FPG, ADA, Uric Acid and HbA1c were also highly significant (p<0.05) (Table 5) which concludes that uric acid as a predictor of oxidative stress become significant as the duration of diabetes increases while ADA is statistically significant even during early onset of diabetes mellitus (Table-5).

DISCUSSION

Diabetes Mellitus comprises a group of common metabolic disorders that share the phenotype of hyperglycemia and results from a defect in insulin secretion, insulin action or both. Insulin deficiency in turn leads to chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism.[17] Oxidative stress plays a major role in cellular injury from hyperglycemia. High glucose level can lead to free radical production. Weak defense system of the body becomes unable to counteract the enhanced reactive oxygen species generation and as a result condition of imbalance between ROS and their protection occurs which leads to domination of the condition of oxidative stress.[18] Physiological roles of ADA can be seen in connection with adenosine whose concentration can be modulated by enzymatic action of ADA. Immunological disturbances in type 2 diabetic individuals have an association with cell mediated responses. Adenosine deaminase, an enzyme distributed in human tissues, was considered as a good marker of cell mediated immunity.[8] Nitric oxide (NO) is the major endothelium-derived relaxing factor associated with oxidative stress and insulin resistance. Uric acid impairs endothelial function and enhances nitric synthase deficiency, which reduces NO, a known mechanism for inducing insulin resistance.[19] Also, Serum Uric Acid has been found to be associated with cardiovascular disease in adults with or without impaired glucose tolerance.[20] According to Gitanjali G

et al. ADA has got a role in increasing lipid peroxidation by reactive oxygen species generation, as they observed positive correlation of ADA with Malondialdehyde (MDA) levels.[11] Mohd Nadeem et al., concluded in their study that uric acid and MDA level are increase in diabetic patients in relation to HbA1C .[21] Yuliang Cui in 2016 observed that in newly diagnosed type 2 diabetes patients the correlation between uric acid and HbA1c most likely relies on insulin levels and they found negative correlation between uric acid and HbA1cl in newly diagnosed type 2 diabetes patients[22]. These findings are similar to the Rusdiana et al., who found no significant association between HbA1c and uric acid levels in type 2 diabetes patients at primary health clinic at Indonesia[23]. Anju Gill et al. concluded in their study that uric acid serves as a potential biomarker of the glucose metabolism.[24] In hyperuricemia or gout, recommended low purine diets are often high in carbohydrate and saturated fat. These macronutrients are associated with an increased risk of the insulin resistance syndrome and associated major consequences.[25,26,27] Furthermore, these macronutrients tend to lead to higher serum insulin levels, which are known to reduce renal excretion of urate thus potentially further increasing the serum uric acid level.[28,29] Melvin R Hayden and Suresh C Tyagi in 2004 reviewed uric acid in metabolic syndrome, type 2DM, and the cardiovascular atherosclerotic afflicted patients and they concluded hyperuricemia should alert the clinician to an overall increased risk of cardiovascular disease. They also concluded that in type 2 DM hyperuricemia is acting through obesity and insulin resistance.[30] Fang J et al. observed that chronic high UA concentrations are associated to increased risk for CAD, acute elevations seem to provide antioxidant protection.[31] Adenosine potentiates insulin and contraction stimulated glucose transport in skeletal muscles by enhancing the increase in GLUT-4 at the cell surface and raised the possibility of decreased adenosine production or action by increased level of adenosine deaminase could play a causative role in insulin resistance.[32]

In meta-analysis derived from 8 prospective cohort studies, serum uric acid was associated with increased risk of developing type 2 DM independent of other risk factors of metabolic syndrome.[33]

In a study conducted by Suchitra mM et al., uric acid and Lp(a) were significantly high and levels of antioxidant vitamins were significantly low in type 2 diabetes mellitus without any cardiovascular complications.[34]

As can be seen from the present study, the serum level of uric acid and ADA was significantly increased in diabetic patients compared to the healthy individuals. This is similar to Mohammed A. Al-Duais suggesting ADA as a good diagnostic marker in Saudi diabetic patients.[35]

Review done by Qing Xiong in 2019 confirmed the positive correlation between uric acid and diabetes mellitus and its chronic complications like diabetic nephropathy through the pathogenesis and clinical studies aspects.[36]

In the present study, serum ADA levels were markedly increased and statistically significant in diabetes mellitus patients in both groups while UA level significant only in diabetes mellitus patients with > 5-year duration which suggest that as after few years uric acid act as a prooxidant and contribute to the oxidative stress as it correlates with increased ADA level also. These findings are in accordance with conclusion of Shashikala Magadi Dasegowda et al., in which ADA, Malonaldehyde (MDA) were elevated and positively correlated with uric acid as a oxidative stress markers.[37]

CONCLUSIONS

It is concluded from the present study that serum ADA and Uric Acid significantly increased in type 2 diabetes mellitus patients. ADA was significant even at early stage of disease with diabetes duration less than a year while serum uric acid was not significant in that group. As diabetes duration increases, hyperuricemia is significant with diabetes group more than 5 years duration. As an oxidative stress marker in metabolic syndrome, in atherosclerosis or in developing chronic complications of diabetes like retinopathy and nephropathy, serum uric acid has been emerged as a parameter to give specific attention in type 2 diabetes mellitus. while ADA has been viewed

as a parameter of interest in type 2 diabetes due to its role in oxidative stress, as a marker of cell mediated immunity along with its effects on insulin by altering levels of adenosine. As global burden of diabetes is increasing, it is imperative to develop appropriate dietary and other lifestyle guidelines taking into account improving hyperuricemia and overall long-term health effects. However, a major limitation of the present study was the small study population, which warrants further cross-sectional studies by using a larger sample size.

REFERENCES

1. Pamela C. Champe, Richard A. Harvey, Denise R. Ferrier. Lippincott's Illustrated reviews, 3rd edition, chapter 25 Diabetes Mellitus, Page no.336.

2. Rajendra P., Viswanathan M. Epidemiology of type 2 diabetes in India, Indian Journal of Ophthalmology, 2021; 69(11): 2932–2938.

3. Carl A. burtis, Edward R. Ashwood, David E. Bruns Tietz Fundamentals of Clinical Chemistry, Sixth Edition, chapter 22,Creatinine, Urea and Uric Acid, Page 368,370

4. Hadeel Ahmed Shawki, Rasha Elzehery2 · Maha Shahin, Ekbal M. Abo-hashem Magdy M. Youssef, Evaluation of some oxidative markers in diabetes and diabetic retinopathy. Diabetology International 2021; 12:108–117

5. Tassone EJ, Presta I, Sciacqua A, Rotundo M, Di Cello S et al. Uric acid promotes endothelial dysfunction: A new molecular model of insulin resistance. Eur J Clin Invest 2011; 41: 81-82.

6. Kodama S, Saito K, Yachi Y, Asumi M, Sugawara A et al. Association between serum uric acid and development of type 2 diabetes. Diabetes Care 2009; 32: 1737-1742. doi:10.2337/dc09-0288. PubMed: 19549729.

7. Lv Q, Meng XF, He FF, Chen S, Su H et al. High serum uric Acid and increased risk of type 2 diabetes: a systemic review and meta-analysis of prospective cohort studies. PLOS ONE 8 2013; e56864. doi:10.1371/journal.pone.0056864. PubMed: 23437258.

8. Gohel Mg, Sirajwala Hb, Kalaria Tr, Kamariya Cp. A Study of Serum Adenosine DeaminaseLevel in Patients with Type 2 Diabetes Mellitus and its correlation with glycemic control.International Journal Of Medical and Applied Sciences 2013;2(3):259-67

9. Seppo Lehto, Leo Niskanen, Tapani Ro¨nnemaa, Markku Laakso, Serum Uric Acid Is a Strong Predictor of Stroke in Patients With Non–Insulin-Dependent Diabetes Mellitus; *Stroke*. 1998;29:635-639

10. H. K. Choi and E. S. Ford, Haemoglobin A1c, fasting glucose, serum C-peptide and insulin resistance in relation to serum uric acid levels—the Third National Health and Nutrition Examination Survey, Rheumatology 2008;47:713–717

11. Gitanjali G, Sudeep G, Neerja, Mili G, Deepak A, Priyanka S. The Effect of Hyperglycaemia on some biochemical parameters in Diabetes Mellitus. Journal of Clinical and Diagnostic Research. 2010;4:3181-86

12. Siddiq SS, Ahmad J, Islam N, Ashraf SMK, Mishra SP. A study on the modulation of adenosine deaminase (ADA) activity in monocytes of type-2 diabetic patients by antioxidants. JIACM 2011; 13(1): 18-21

13. Carl A. burtis, Edward R. Ashwood, David E. Bruns. Tietz textbook of clinical chemistry and molecular diagnostics 4th edition, ch. 25 carbohydrates, pg. 870

14. Giusti G, Galanti B. Soc. Ital. Biol. Sper 1966; 42: 1316-1320

15. Carl A. burtis, Edward R. Ashwood, David E. Bruns. Tietz textbook of clinical chemistry and molecular diagnostics 4th edition, ch. 24 kidney function test, pg. 807

16. Carl A. burtis, Edward R. Ashwood, David E. Bruns. Tietz Fundamentals of Clinical Chemistry, Sixth Edition, chapter 22 cabohydtares, methods for determaination of HbA1c, pg. 396

17. Richard Kahn, Duke St, Alexandria. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 1997;20(7):1183-97

18. Halliwell B, Gutteridge J Free Radicals in Biology and Medicine. Oxford University Press, New York, NY, USA. 2007.

19. Li C, Hsieh MC, Chang SJ. Metabolic syndrome, diabetes, and hyperuricemia. Curr Opin Rheumatol. 2013;25(2):210–6.

20. Wijnands JM, et al. The cross-sectional association between uric acid and atherosclerosis and the role of low-grade inflammation: the CODAM study. Rheumatology (Oxford). 2014;53(11):2053–62.

21. Mohd Nadeem, Balwinder Kaur, Sachin Agarwal Comparison Of Oxidative Stress Level In Diabetes Mellitus As Compare To Normal Individual. Journal of Cardiovascular Disease Research. 2022; 13(7):892-897

22. Yuliang Cui, Hemei Bu, Xianghua Ma, Sha Zhao, Xiaona Li, and Shan Lu. The Relation between Serum Uric Acid and HbA1c Is Dependent upon Hyperinsulinemia in Patients with Newly Diagnosed Type 2 Diabetes Mellitus. Journal of Diabetes Research; Volume 2016, Article ID 7184123, 6 pages

23. Rusdiana, Sri Suryani Widjaja et al., Association between Haemoglobin A1c and Uric Acid Levels among Patients with Diabetes Mellitus Type 2 at a Primary Health Care Clinic in North Sumatera, Indonesia. Open Acess Macedonian Journal of Medical science 2018; 6(9) : 1633-1635

24. Anju Gill, Shhiba Kukreja, Naresh Malhotra, Namrata Chhabra. Correlation of Serum Insulin and the Serum Uric Acid Levels with the Glycated Haemoglobin Levels in Patients of Type 2 Diabetes Mellitus. Journal of Clinical and Diagnostic Research. 2013;7(7):1295-1297

25. Fam AG. Gout in the elderly. Clinical presentation and treatment. Drugs & Aging. 1998;13:229-243.

26. Dessein PH, Shipton EA, Stanwix AE, Joffe BI, Ramokgadi J. Beneficial effects of weight loss associated with moderate calorie/carbohydrate restriction, and increased proportional intake of protein and unsaturated fat on serum urate and lipoprotein levels in gout: a pilot study. Ann Rheum Dis. 2000;59:539-543.

27. Parillo M, Rivellese AA, Ciardullo AV, et al. A high-monounsaturatedfat/ low-carbohydrate diet improves peripheral insulin sensitivity in noninsulin-dependent diabetic patients. Metabolism. 1992;41:1373-1378.

28. Ter Maaten JC, Voorburg A, Heine RJ, Ter Wee PM, Donker AJ, Gans RO. Renal handling of urate and sodium during acute physiological hyperinsulinaemia in healthy subjects. Clin Sci (Lond). 1997;92:51-58.

29. Muscelli E, Natali A, Bianchi S, et al. Effect of insulin on renal sodium and uric acid handling in essential hypertension. Am J Hypertens.1996;9:746-752.

30. Melvin R Hayde and Suresh C Tyagi, Uric acid: A new look at an old risk marker for cardiovascular disease, metabolic syndrome, and type 2 diabetes mellitus: The urate redox shuttle; Nutrition & Metabolism 2004, 1:10

31. Fang J: Alderman MH: Serum uric acid and cardiovascular mortality the NHANES I epidemiologic follow-up study, 1971-1992. National Health and Nutrition Examination Survey. Jama, 2000; 283: 2404-2410. 10.1001/jama.283.18.2404.

32.Dong-Ho Han, Polly A. Hansen, Lorraine A. Nolte, and John O. Holloszy. Removal of Adenosine Decreases the responsiveness of muscle glucose transport to insulin and contractions. Diabetes 1998;47:1671–1675.

33. Qin Lv., Xian-Fang Meng et al., High Serum Uric Acid and Increased Risk of Type 2 Diabetes: A Systemic Review and Meta-Analysis of Prospective Cohort Studies.www.plosone.org; 2013 ;8 (2) e56864

34. Suchitra mM , Pallavi m, Shivaprasad p, Sachan, et al. Lp (a), Uric Acid, Oxidants and Antioxidant Vitamins in Type 2 Diabetic Patients without Cardiovascular Complications. Journal of Clinical and Diagnostic Research. 2011 November (Suppl-1), Vol-5(6): 1161-1164

35. Mohammed A. Al-Duais et al., Diagnostic Value of Serum Adenosine Deaminase in Type II Saudi Diabetic Patients. Adv Diabetes Endocrinol 2015;1(1): 5.

36. Qing Xiong ,Jie Liu , and Yancheng Xu. Effects of Uric Acid on Diabetes Mellitus and Its Chronic Complications. International Journal of Endocrinology; Volume 2019, Article ID 9691345, 8 pages

37. Shashikala Magadi Dasegowda , Ashok Kumar Jeppu et al., Serum uric acid and its relation to adenosine deaminase, lipid profile and oxidative stress in Diabetes Mellitus type 2. Journal of Investigational Biochemistry; 2016;5(2):32-36

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