Original Article

Association of Serum Uric Acid and Lipid Profile in Adults at a Tertiary Level Hospital in Bangladesh

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Abstract

Background: *While some studies have found a connection between increased uric acid and metabolic syndrome, nothing is currently known about the interaction between serum uric acid (SUA) and lipid profile. The purpose of this study was to evaluate the association between SUA and lipid profile in Bangladeshi adults as a whole.* ***Materials and Methods:*** *In this cross-sectional study, a total of 140 blood samples were collected from apparently healthy adults aged >18 years. SUA, lipid profile, liver enzymes and other biochemical markers were measured in the collected samples by using standard methods. All statistical analyses were performed by using SPSS version22.0 software and p<0.05 was considered statistically significant.* ***Results:*** *A total of 140 subjects were selected. Among them 70 were male and 70 were female. SUA showed significant positive correlation between SUA and TC, TG, LDL-C while negative correlation between SUA and HDL-C among all subjects (p<0.001). It also revealed that SUA, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT) levels were significantly higher (p<0.001) in male than female group. Pearson's correlation analysis showed that in all subjects there were significant positive correlation between SUA and serum ALT, AST & GGT (p<0.001).* ***Conclusion:*** *SUA is positively correlated with TC, TG and LDL-C in adults. More prospective studies are needed to clarify the complex relationship between SUA and lipid profile in the general population.*

Keywords: Serum uric acid, Total cholesterol, Triglyceride, LDL cholesterol, HDL cholesterol.

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Introduction

Serum uric acid (SUA) is the major end product of the purine metabolism and the level of SUA is maintained by the balance between SUA production and excretion.[1] Intracellularly uric acid can act as a pro-oxidant inducing the release of inflammatory mediators and growth factors.[2] Uric acid has been shown to contribute to lipoprotein oxidation and inflammation which are thought to play vital roles in the development and progression of nonalcoholic fatty liver disease (NAFLD).[3] Hyperuricemia has been linked to both metabolic syndrome (MetS) and cardiovascular disease.[4] The SUA was increased in most NAFLD patients which was an independent risk factor for NAFLD. Therefore, increased SUA may play the role of linking NAFLD with MetS.[5] Dyslipidemia has long been understood to be a major metabolic event that raises the risk of cardiovascular disease (CVD) and atherosclerosis. It manifests as a change in the blood's lipoprotein concentrations. In Bangladesh, the levels of triglycerides, LDL cholesterol, and mean cholesterol are all rising. It has long been known that dyslipidemia is a major metabolic event that is associated with atherogenicity and cardiovascular disease (CVD). It can show up as a change in the concentrations of lipoprotein in plasma.[6]

Patients with type 2 diabetes mellitus (T2DM) are thought to be at increased risk for renal problems due to elevated serum uric acid levels. In type 2 diabetes, dyslipidemia poses a general risk for

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cardiovascular problems. In T2DM patients, a greater amount of SUA is thought to be a risk factor for kidney disorders.[7] A number of cardiovascular disorders have been linked to abnormal levels of low-density lipoprotein cholesterols (LDL-C) and very-low-density lipoprotein cholesterols (VLDL-C). Cardiovascular disorders of various kinds are the most frequent consequences among people with type 2 diabetes. Therefore, in T2DM patients, dyslipidemia, frequently raised triglyceride (TG) levels, and low levels of high-density lipoprotein cholesterol (HDL-C) are significant issues.[8] Evidence suggests that elevated SUA levels and increased activity of liver enzymes gamma-glutamyl transferase (GGT) and alanine transaminase (ALT) and to a lesser extent aspartate transaminase (AST) are also associated with the development of MetS and NAFLD. Hyperuricemia is quite prevalent worldwide; rates of 35.1% in the Seychelles for men and 8.7% for women, 10.6% in Thailand, 7.2% for men and 0.04% for women in England and Scotland, 11.2% in the USA, and 8.4% in Saudi Arabia have all been documented. It is unclear how hyperuricemia and dyslipidemia are related.[9–13]

The overall population now experiences hyperuricemia more frequently due to changes in modern lifestyle. Uric acid crystals start to develop on the artery wall when uric acid levels reach beyond 7 mg/dl, a condition known as hyper saturation. Conversely, men and women are valued differently. Monosodium urate binds to plasma IgG. Platelets become activated when the monosodium urate-IgG combination is recognized by the Fc receptor on the platelets. Through this process, blood coagulation is induced. Cytokine secretion is the cause of atherosclerosis progression.[14] Elevated levels of blood uric acid have been associated with elevated insulin levels, also referred to as hyperinsulinemia, reduced physical activity, excessive alcohol use, hypertension, elevated body mass index (BMI) and decreased HDL cholesterol levels.[15] These traits are all associated with an increased risk of stroke and coronary artery disease (CAD). Because of this, there is disagreement on whether uric acid causes the pathologic processes observed in these disorders or if it is merely a co-existing marker.[16] Numerous investigations have shown that, in patients with coronary artery disease (CAD), hyperuricemia may be utilized as an independent predictor of death after standard risk indicators were taken into consideration.[17] This study was conducted as an attempt to find association between uric acid levels and lipid profile in Bangladeshi adults.

Materials and Methods

Study settings and study population: This cross-sectional analytical study was conducted from 1st March, 2019 to 29th February, 2020 at the Department of Biochemistry, Sir Salimullah Medical College, Dhaka, Bangladesh. The study was conducted on 140 subjects (70 males and 70 females). Apparently healthy people of 30 to 59 years were included in the study. These subjects were selected from the attendants accompanying the patients attending the outpatient department of Sir Salimullah Medical College and Mitford Hospital. Study population included both male and female categorized into four quartiles including hyperuricemia on the basis of SUA level. Inclusion criteria: (i) Healthy adults with age range of 30- 59 years (ii) Both genders. Exclusion criteria: (i) Subjects with DM and renal failure. (ii) Those taking anti-hypertensive, anti-diabetic, lipid-lowering and hypouricemic drugs. (iii) Chronic liver disease such as cirrhosis, liver cancer, viral hepatitis, autoimmune hepatitis and taking hepatotoxic drugs and (iv) Alcoholism.

Anthropometric data collection: Using a measuring device, height was measured (shoes off) and recorded to the closest few centimeters. Body

weight was assessed without shoes and in light clothing. Weight in kilograms divided by height in meters squared yielded the body mass index (BMI). After at least five minutes of rest in a quiet room, blood pressure was taken three times at two-minute intervals while sitting down using a manual sphygmomanometer. All analyses were performed using the computed mean of the three blood pressure readings. Each patient was interviewed prior to the collection of specimens, and pertinent information was methodically documented in a standard pre-designed data sheet. The data was then amended and verified.

Study procedure: After a standard baseline biochemical investigation, a physical examination, and a history, these patients were recruited. Following appropriate counseling, each participant received a detailed explanation of the study's purpose, goals, risks, and methodology. Candidates were only sought out voluntarily to participate in the study. The participants were allowed to leave the research at any time and written informed permission was obtained from each subject. Socio-demographic as well as other relevant data were taken and recorded in the data collection sheet with a prefixed questionnaire. A blood sample was collected for biochemical variables to be measured.

Blood collection and laboratory analysis: Each participant's overnight fasting blood sample, which included about 5 mL, was taken for biochemical evaluations. Serum uric acid (SUA), fasting plasma glucose (FPG), triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), liver enzymes - alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were all measured in the blood biochemical studies. Serum kinetic techniques were used to determine the liver enzyme activity, and other biochemical parameters were examined by conventional colorimetric techniques. A biochemistry analyzer was used to measure the biochemical parameters. Biochemical tests were done in the Biochemistry laboratory of Sir Salimullah Medical College, Dhaka.

Operational definitions:

- **Elevated liver enzymes** defined as ALT >45 U/L in male and >34 U/L in female; AST >35 U/L in male and >31 U/L in female; GGT >55 U/L in male and >38 U/L in female.[18]
- **Hyperuricemia** was defined according to sex-specific SUA levels: SUA >7.0 mg/dL for male and >6.0 mg/dL for female.[19]
- **Lipid profile** cutoff level of TC, TG, HDL-C and LDL-C were < 200 mg/dl, < 150 mg/dl, > 40 mg/dl and < 100 mg/dl, respectively.[20]

Statistical analysis: Continuous variables were expressed as mean values and standard deviation (SD), whereas categorical variables were described as frequencies and percentages. SUA levels were divided into quartiles including hyperuricemia. Statistical methods followed were unpaired students' t-test, analysis of variance (ANOVA) test where indicated. Bonferroni test was performed to show the difference in between different categories of SUA level. Pearson's correlation test was performed to analyze the relation between SUA level and lipid profile. All statistical analyses were performed by using SPSS version 22.0 software and $p<0.05$ was considered statistically significant.

Results

A total of 140 participants were chosen for this investigation. There were 70 males and 70

females among them. The study individuals' baseline characteristics are shown in Table I. Male average age (±SD) was 44.43±7.32 years, while female average was 42.80±6.92 years. Additionally, it showed that there was no age-related statistically significant difference between the two groups. Regarding BMI, there was no significant difference found between the two cohorts. Males had a considerably higher mean (±SD) SBP in mmHg than females (130.83±12.08 vs. 125.71±13.89, p<0.05). The average DBP in mmHg (87.50±9.66 against 82.93±9.80, p<0.01) showed a significant difference between the male and female genders.

Table II showed mean±SD of the biochemical parameters. Analysis of lipid profile showed that TC (p<0.05) and LDL-C (p<0.05) level were significantly higher in male than female subjects whereas HDL-C (p<0.01) was significantly lower in male than female group and also serum uric acid (SUA), ALT, AST and GGT levels were significantly (p<0.001) higher in male than female group. How-

Table I: Baseline characteristics of study subjects (n=140)

Variables	All subjects (n=140)	Male (n=70)	Female (n=70)	*p*-value
Age (years)	43.62±7.15	44.43±7.32	42.80±6.92	0.175
BMI (kg/m^2)	24.11±2.86	24.51±2.87	23.71±2.81	0.099
SBP (mmHg)	128.27±13.22	130.83±12.08	125.71±13.89	<0.05
DBP (mmHg)	85.21±9.96	87.50±9.66	82.93±9.80	<0.01

Results were expressed as mean±SD.
Unpaired student's t-test was performed to compare between group means.

Table II: Biochemical parameters of study subjects (n=140)

Biochemical parameters	All subjects (n=140)	Male (n=70)	Female (n=70)	*p*-value
FPG (mmol/L)	5.34±1.11	5.45±1.16	5.22±1.04	0.228
*SUA (mg/dl)	4.94±1.35	5.46±1.33	4.49±1.32	<0.001
*TC (mg/dl)	193.86±21.97	198.30±23.04	189.43±20.03	<0.05
*TG (mg/dl)	157.01±26.09	160.14±21.15	153.89±30.06	0.157
*HDL-C (mg/dl)	39.93±4.38	38.90±4.08	40.96±4.46	<0.01
*LDL-C (mg/dl)	125.80±12.60	128.09±13.21	123.51±11.61	<0.05
ALT (U/L)	29.51±1.33	33.88±1.32	25.70±1.26	<0.001
AST (U/L)	27.54±1.32	31.62±1.29	24.55±1.27	<0.001
GGT (U/L)	35.48±1.46	43.65±1.42	29.51±1.34	<0.001

Results were expressed as mean±SD and *geometric mean±SD.
Unpaired student's t-test was performed to compare between group means.

ever, in cases of TG and FPG no significant difference was observed in between groups.

Table III showed the characteristics of study subjects categorized by serum uric acid (SUA) level. Participants in the higher quartiles of SUA and hyperuricemia showed an increasing pattern of age as well as BMI, SBP and DBP. Serum biochemical parameters such as TC, TG, LDL-C, ALT, AST, GGT and FPG all intended to increase from lowest to highest quartiles of SUA and hyperuricemic subgroup while HDL-C intended to decrease from lowest to highest quartiles of SUA and hyperuricemic subgroup (p<0.001).

Table III: Characteristics of subjects categorized by SUA level in all subjects (n=140)

Variables	Quartiles of normal serum uric acid (SUA)				Hyperuricemia (n=18)	*p*-value
	Q1 (n=26)	Q2 (n=32)	Q3 (n=34)	Q4 (n=30)		
*SUA (mg/dl)	3.34±1.14	4.20±1.08	4.97±1.09	6.05±1.08	8.28±1.15	<0.001
Age (yrs)	37.54±5.85	39.75±5.61	44.06±5.60	48.50±5.53	50.33±4.70	<0.001
BMI (kg/m^2)	21.47±2.73	23.36±2.34	24.15±2.42	25.21±1.98	27.38±1.63	<0.001
SBP(mmHg)	118.65±12.37	122.19±10.77	127.50±12.51	135.93±7.45	141.67±9.39	<0.001
DBP(mmHg)	77.69±8.63	80.94±8.93	87.79±7.41	88.50±8.00	93.33±10.85	<0.001
FPG (mmol/L)	4.67±0.96	5.05±0.99	5.17±0.75	5.55±0.65	6.78±1.37	<0.001
*TC (mg/dl)	178.42±21.50	183.47±14.89	190.18±12.88	205.60±14.75	222.06±22.10	<0.001
*TG (mg/dl)	135.85±12.28	146.13±21.69	160.29±32.52	169.37±15.41	180.17±16.12	<0.001
*HDL-C (mg/dl)	42.73±3.69	41.81±3.37	40.50±4.16	37.43±3.65	35.61±3.11	<0.001
*LDL-C (mg/dl)	115.69±11.83	120.97±10.61	124.32±9.16	133.60±8.60	138.78±10.36	<0.001
ALT (U/L)	23.44±1.34	26.91±1.29	29.51±1.26	33.88±1.20	39.81±1.16	<0.001
AST (U/L)	21.88±1.38	25.70±1.25	27.54±1.27	32.36±1.16	37.15±1.16	<0.001
GGT (U/L)	25.12±1.49	31.62±1.41	36.31±1.33	44.67±1.26	50.11±1.22	<0.001

Results were expressed as mean±SD and *geometric mean±SD. ANOVA test was performed to compare all the variables between normal SUA and hyperuricemia.

In table IV Pearson's correlation analysis showed that significant positive correlation between SUA

and TC, TG, LDL-C while negative correlation between SUA and HDL-C among all subjects (p<0.001). These correlations were also significant among male and female subjects. And also in all subjects there were significant positive correlation between SUA and serum ALT, AST, GGT (p<0.001).

increasing trend in levels of TC, TG, LDL-C, FPG as well as age, BMI, SBP, DBP except HDL-C which showed decreasing trend with increasing SUA level. Nearly similar pattern of observation was reported by two studies.[5,22] Following stratification based on the SUA level quartiles, it became clear that participants of both genders had higher

Table IV: Correlation of SUA (mg/dl) with TC, TG, HDL-C, LDL-C in study subjects (n=140)

Parameters	All subjects (n=140)		Male (n=70)		Female (n=70)	
	r value	*p*-value	r value	*p*-value	r value	*p*-value
TC (mg/dl)	+0.621	<0.001	+0.640	<0.001	+0.541	<0.001
TG (mg/dl)	+0.534	<0.001	+0.696	<0.001	+0.420	<0.001
HDL-C(mg/dl)	-0.549	<0.001	-0.446	<0.001	-0.617	<0.001
LDL-C (mg/dl)	+0.592	<0.001	+0.615	<0.001	+0.513	<0.001
ALT(U/L)	+0.656	<0.001	+0.580	<0.001	+0.633	<0.001
AST(U/L)	+0.678	<0.001	+0.607	<0.001	+0.653	<0.001
GGT(U/L)	+0.697	<0.001	+0.613	<0.001	+0.706	<0.001

Data were log transformed.
Correlations were determined by pearson's correlation co-efficient test.

Discussion

In this study it was observed that there was no significant age difference between male and female subjects. However, it was not consistent with the study done by Yang[21] who found a significant age difference between two groups. The study showed that BMI did not differ in between two groups. Similar results were observed by a Chinese study.[21] In this study, mean (±SD) SBP and DBP in mmHg of male differed to some extent from those of female (130.83±12.08 vs 125.71±13.89 and 87.50±9.66 vs 82.93±9.80, p<0.01). Similar observation was found in other studies.[5,21] It was evident from the study that participants with hyperuricemia as well as those in the highest quartile (Q4) of SUA within reference range had elevated lipid profile (TC, TG, LDL-C). The data of this study also revealed that there is a stepwise increase in lipid profile with increasing levels of SUA even within the reference range. Participants with increasing quartiles of SUA and hyperuricemia showed an

percentages of TC, TG, and LDL fat profiles as well as higher uric acid quartiles overall and in the hyperuricemic subgroup. Several researchers noticed almost identical results.[5,21] In line with previous research, the current study found that SUA was inversely connected with HDL-C in both genders and significantly and positively correlated with TC, TG, LDL-C, as well as ALT, AST, and GGT.[23] Additionally, our results suggested that a higher SUA is connected to a lipid profile on its own.

According to Zhang[24], elevated xanthine oxidoreductase activity might cause an increase in uric acid production, which can hasten the onset of nonalcoholic fatty liver disease (NAFLD). By encouraging the over-expression of pro-lipogenic enzymes such as sterol regulatory element binding proteins[25], elevated uric acid causes the buildup of triglycerides. Research indicates that reactive oxygen species are produced in tandem with the uric acid that xanthine oxidoreductase catalyzes. Thus, NAFLD development is caused by oxidative stress mediated by xanthine oxidoreductase.[26]

However, it has been reported that SUA is significantly associated with hypertension, obesity, CVD, hypertriglyceridemia and hyperglycemia which may increase the risk of MetS.[27] Moreover, SUA has been considered as a marker of oxidative stress associated with CVD[28] and MetS. MetS is therefore thought to have a significant impact on the onset or course of NAFLD. Increased blood uric acid levels have also been linked to the onset of nonalcoholic fatty liver disease (NAFLD), which can result in cirrhosis. According to certain research, uric acid levels and lipid profiles are highly correlated, and uric acid levels may be impacted by factors such as age, gender, smoking, alcohol use, obesity, and insulin resistance.[29] Our findings are supported by the study conducted by Baliarsingh[30], which shown that hypertriglyceridemia is significantly elevated when serum uric acid levels rise.

There is enough evidence to conclude that cardiovascular illnesses and hyperuricemia are related.[31–34] In addition to being linked to an increased risk of oxidative stress and the production of free radicals, hyperuricemia predisposes people to the development of hypertension and may eventually be the precursor to cardiovascular disease.[35] The study using animal models has demonstrated that uric acid significantly increases MCP-1 expression and release, macrophage infiltration, and the expression of the proinflammatory cytokine TNF-α in vascular smooth muscles. These events can ultimately result in smooth muscle atherosclerosis.[36–38] There are additional factors that raise a patient's risk of CVD in hyperuricemic individuals. Bendek presented one of the first studies demonstrating hypertriglyceridemia in hyperuricemic individuals, and further research has confirmed this correlation.[39] There is an inverse association between uric acid and HDL, according to several research. HDL negatively correlate with hyperuricemia led to a drop in HDL, which in turn increases atherosclerosis and ultimately put people at risk for CVD. LDL cholesterol is always regarded as bad cholesterol, and an increase in LDL causes atherosclerosis, which can results a variety of cardiovascular problems, including myocardial infarction and angina.[40–42]

In our study, it can be concluded that almost all the variables except HDL-C are increased with the increment of SUA level and hyperuricemia. Increased SUA is associated with elevated lipid profile (TC, TG, LDL-C) and also liver enzymes (ALT, AST and GGT) in adults and this association is independent of other confounding factors. Subjects in higher quartiles of SUA and hyperuricemia showed an increasing pattern of age with significantly higher BMI, SBP, DBP, FPG, TC, TG, LDL-C, ALT, AST, GGT and lower HDL-C. The variation of these parameters based on quartiles of SUA and hyperuricemia were almost similar in both genders. Pearson's correlation analysis showed positive correlation of SUA with TC, TG, LDL-C, ALT, AST, GGT and negative correlation with HDL-C among all male as well female study subjects.

Limitations

The present study shows an important correlation between lipid profiles and SUA in Bangladeshi adults. Moreover, adult Bangladeshis may have a decreased risk of associated cardiovascular disease if they do not have dyslipidemia or hyperuricemia. Further investigation that takes factors such as lifestyle, diabetes, and hypertension is needed to have a more thorough understanding of the study outcome.

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Conflict of interest: Authors have no conflict of interest to declare.

Ethical approval: This study was approved by the Institutional Ethics Committee of Sir Salimullah Medical College, Dhaka (SSMC/2019/194). All participants were informed about the study and they gave their written consent before inclusion in the study.

References

1. Hediger MA, Johnson RJ, Miyazaki H et al. Molecular physiology of urate transport. Physiology. 2005 Apr;20(2):125-133.

2. Kang DH, Han L, Ouyang X et al. Uric acid causes vascular smooth muscle cell proliferation by entering cells via a functional urate transporter. Am J Nephrol. 2005 Sep-Oct;25(5):425-433.

3. Baldwin W, McRae S, Marek G et al. Hyperuricemia as a mediator of the proinflammatory endocrine imbalance in the adipose tissue in a murine model of the metabolic syndrome. Diabetes. 2011 Apr 1;60(4):1258-1269.

4. Gagliardi AC, Miname MH, Santos RD. Uric acid: a marker of increased cardiovascular risk. Atherosclerosis. 2009 Jan 1;202(1):11-17.

5. Chen S, Guo X, Yu S et al. Association between serum uric acid and elevated alanine aminotransferase in the general population. Int J Environ Res Public Health. 2016 Aug 24;13(9):841.

6. Kumar S, Mondal H, Lata M et al. Correlation of serum uric acid with lipid profile in patients with type 2 diabetes mellitus with normal creatinine level: Report from a tertiary care hospital in India. J Family Med Prim Care. 2022 Jun 1;11(6):3066 -3070.

7. Obermayr RP, Temml C, Gutjahr G et al. Elevated uric acid increases the risk for kidney disease. J Am Soc Nephrol. 2008 Dec;19(12): 2407-2413.

8. Battisti WP, Palmisano J, Keane WE. Dyslipidemia in patients with type 2 diabetes. relationships between lipids, kidney disease and cardiovascular disease. Clin Chem Lab Med. 2003 Sep;41(9):1174-1181.

9. Al-Meshaweh AF, Jafar Y, Asem M et al. Determinants of blood uric acid levels in a dyslipidemic Arab population. Med Princ Pract. 2012;21(3):209-216.

10. Villegas R, Xiang YB, Cai Q et al. Prevalence and determinants of hyperuricemia in middle-aged, urban Chinese men. Metab Syndr Relat Disord. 2010 Jun;8(3):263-370.

11. Lohsoonthorn V, Dhanamun B, Williams MA. Prevalence of hyperuricemia and its relationship with metabolic syndrome in Thai adults receiving annual health exams. Arch Med Res. 2006 Oct;37(7):883-889.

12. Sturge RA, Scott JT, Kennedy AC et al. Serum uric acid in England and Scotland. Ann Rheum Dis. 1977 Oct;36(5):420-427.

13. Schmidt MI, Watson RL, Duncan BB et al. Clustering of dyslipidemia, hyperuricemia, diabetes, and hypertension and its association with fasting insulin and central and overall obesity in a general population. Atherosclerosis Risk in Communities Study Investigators. Metabolism. 1996 Jun;45(6):699-706.

14. Agete TH, Eshetu NG. Factors associated with atherogenic dyslipidemia among hypertensive patients at southern Ethiopia. Int. J. Med. Med. Sci. 2018 Jul 31;10(7):86-93.

15. Yu Y, Fang W, Wang D et al. Contribution of Nontraditional Lipid Profiles to Hyperuricemia in a Hypertensive Population: Findings from the China Hypertension Registry Study. [Incomplete peer reviewed preprint manuscript]

16. Athyros VG, Mikhailidis DP, Papageorgiou AA et al. Targeting vascular risk in patients with metabolic syndrome but without diabetes. Metabolism. 2005 Aug 1;54(8):1065-1074.

17. Rathore V, Singh N, Rastogi P et al. Lipid profile and its correlation with C-reactive protein in patients of acute myocardial infarction. Int J Res Med Sci. 2017 May;5(5):2182-2186.

18. Lopez J. Carl A. Burtis and David E. Bruns: Tietz Fundamentals of Clinical Chemistry and Molecular Diagnostics, 7th ed: Elsevier, Amsterdam, 1075 pp, ISBN 978-1-4557-4165-6. Indian J Clin Biochem. 2015 Apr;30(2):243.

19. Lamb EJ, Path FR, Price CP. 21 Kidney Function Tests—Creatinine, Urea, and Uric Acid. Tietz Fundamentals of Clinical Chemistry and Molecular Diagnostics-E-Book. 2014 Feb 26:364.

20. Expert Panel on Detection E. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III). Jama. 2001 May 16;285(19):2486-2497.

21. Yang H, Li D, Song X et al. Joint associations of serum uric acid and ALT with NAFLD in elderly men and women: a Chinese cross-sectional study. J Transl Med. 2018 Oct 17;16(1):285.

22. Zhou Z, Song K, Qiu J et al. Associations between serum uric acid and the remission of non-alcoholic fatty liver disease in Chinese males. PLoS One. 2016 Nov 11;11(11): e0166072.

23. Nakamura K, Sakurai M, Miura K et al. Serum gamma-glutamyltransferase and the risk of hyperuricemia: a 6-year prospective study in Japanese men. Horm Metab Res. 2012 Dec;44(13):966-974.

24. Zhang J, Xu C, Zhao Y et al. The significance of serum xanthine oxidoreductase in patients with nonalcoholic fatty liver disease. Clin Lab. 2014;60(8):1301-1307.

25. -Choi YJ, Shin HS, Choi HS et al. Uric acid induces fat accumulation via generation of endoplasmic reticulum stress and SREBP-1c activation in hepatocytes. Lab Invest. 2014 Oct;94(10):1114-1125.

26. Baskol G, Baskol M, Kocer D. Oxidative stress and antioxidant defenses in serum of patients with non-alcoholic steatohepatitis. Clin Biochem. 2007 Jul;40(11):776-780.

27. Conen D, Wietlisbach V, Bovet P et al. Prevalence of hyperuricemia and relation of serum uric acid with cardiovascular risk factors in a developing country. BMC public health. 2004 Dec; 4:1-9.

28. Marchesini G, Brizi M, Bianchi G et al. Nonalcoholic fatty liver disease: a feature of the metabolic syndrome. Diabetes. 2001 Aug 1;50(8):1844-1850.

29. Zhao LJ, Zhao D, Liu J et al. Association between serum uric acid and triglyceride in a Chinese community. Zhonghua nei ke za zhi. 2005 Sep 1;44(9):664-667.

30. Baliarsingh S, Sharma N. Serum uric acid level is an indicator of total cholesterol and low density lipoprotein cholesterol in men below 45 years in age but not older males. Clin Lab. 2012;58(5-6):545-450.

31. GERTLER MM, GARN SM, LEVINE SA. Serum uric acid in relation to age and physique in health and in coronary heart disease. Ann Intern Med. 1951 Jun;34(6):1421-1431.

32. Verdecchia P, Schillaci G, Reboldi G et al. Relation between serum uric acid and risk of cardiovascular disease in essential hypertension: the PIUMA study. Hypertension. 2000 Dec;36(6):1072-1078.

33. Rahman MA, Islam S, Lubaba MS, Akram A. Study of Uric acid and Serum Creatinine in Diabetic and Non-Diabetic patients in a tertiary hospital, Dhaka. Int. j. adv. multidisc. res. stud. 2022; 2(6):226-228.

34. Kang DH. Potential role of uric Acid as a risk factor for cardiovascular disease. Korean J Intern Med. 2010 Mar;25(1):18-20.

35. Baldwin W, McRae S, Marek G et al. Hyperuricemia as a mediator of the proinflammatory endocrine imbalance in the adipose tissue in a murine model of the metabolic syndrome. Diabetes. 2011 Apr 1;60(4):1258-1269.

36. AshiqurRahman M, Islam S, Rahaman S et al. Assessment the Levels of Serum Ferritin and Some Biochemical Parameters in Type 2 Diabetic Subjects Attending A Tertiary Hospital in Bangladesh. Molecular Mechanism Research. 2023 Dec 13;1(1).

37. Benedek TG. Correlations of serum uric acid and lipid concentrations in normal, gouty, and atherosclerotic men. Ann Intern Med. 1967 May;66(5):851-861.

38. Lin SD, Tsai DH, Hsu SR. Association between serum uric acid level and components of the metabolic syndrome. J Chin Med Assoc. 2006 Nov;69(11):512-516.

39. Perez-Pozo SE, Schold J, Nakagawa T et al. Excessive fructose intake induces the features of metabolic syndrome in healthy adult men: role of uric acid in the hypertensive response. Int J Obes (Lond). 2010 Mar;34(3):454-561.

40. Islam S, AshiqurRahman M, AnamChowdhury S et al. A review: Serum Lipid Profile Status in Cardiovascular Disease. Molecular Mechanism Research. 2023 Dec 13;1(1).

41. Shelmadine B, Bowden RG, Wilson RL et al. The effects of lowering uric acid levels using allopurinol on markers of metabolic syndrome in end-stage renal disease patients: a pilot study. Anadolu Kardiyol Derg. 2009 Oct;9(5):385-389.

42. Islam S, Hossen MA, Rahman MA et al. Serum uric acid level among type-2 diabetes subjects attending in a tertiary hospital of Bangladesh. WJBPHS. 2022;12(1):081-085.