Print ISSN 1110-208X.
Online ISSN 2357-0016

Original article

Serum Uric Acid in Metabolic Associated Fatty Liver Disease in Obese and Lean Patients

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Received: 8 March 2023

Accepted: 25 April 2023

Abstract:

Background: Non-alcoholic fatty liver disease or more recently re-defined metabolic associated fatty liver disease (MAFLD) is regarded as the most relevant liver disease of the twenty-first century, affecting at least one third of the general population. This study aimed to study the association between serum uric acid (SUA) and MAFLD in obese and lean patients. **Methods:** This cross-sectional study was conducted on lean MAFLD patients compared with obese MAFLD patients who attended inpatient and outpatient clinics of Hepatology and Gastroenterology department of Benha University Hospital. Patients were divided into 2 equal groups: Group I: included 45 lean MAFLD patients and Group II: included 45 obese MAFLD patients. All patients were subjected to full history taking, clinical examination, lab investigation, viral markers, lipid profile and imaging. **Results:** Serum uric acid were insignificantly different between both groups (5.42±1.43 in group I and 5.64±1.75 in group II with P-value > 0.05). Serum creatinine was significantly lower in group I compared to group II (P value =0.001). There was a significant positive correlation between CAP score and Albumin (r=0.222, P=0.036), cholesterol (r=0.333, P=0.001) and serum uric acid (r=0.289, P=0.006). There was a significant positive correlation between serum uric acid and cholesterol (r=0.300, P=0.004), LDL (r=0.301, P=0.00) and CAP score (r=0.289, P=0.006). **Conclusion:** Our study supports the rationale for serum uric acid (SUA) being established as another risk factor for metabolic dysfunctions in lean/normal-weight and obese MAFLD.

Keywords: Serum Uric Acid; MAFLD; Obese; Lean Patients.

Introduction

Non-alcoholic or more recently re-defined metabolic associated fatty liver disease (MAFLD) is regarded as the most relevant liver disease of the twenty-first century, affecting at least one third of the general population and it is predicted to become the leading cause of liver transplantation by 2030 (1)The criteria of MAFLD that incorporates 'positive criteria' for the diagnosis, ensures that MAFLD is a clear, distinct entity, These criteria are based on detection of steatosis with one of different modalities (imaging, blood biomarker or histology) with the presence of one of the three criteria, overweight or obesity, type 2 diabetes mellitus or evidence of metabolic abnormalities such as an increased waist circumference and an abnormal lipid or glycemic profile (2).

Serum uric acid (SUA) is the major end product of purine metabolism in humans; and the level of SUA is rigorously controlled by the balance between uric acid production and excretion (3).

Previous studies have shown that SUA level was significantly associated with fatty liver disease and elevated SUA level was an independent risk factor for non-alcoholic fatty liver disease (NAFLD) (4, 5).

To the best of our knowledge, no study has been performed so far to investigate the association of the level of SUA with MAFLD. Therefore, this study aimed to study the association between SUA and MAFLD in obese and lean patients.

Patients and methods

This cross-sectional study was conducted on lean MAFLD patients compared with obese MAFLD patients who attended inpatient and outpatient clinics of Hepatology and Gastroenterology Department of Benha University Hospital over a period of two years from January 2021 to January 2023. The study was done after being approved by the institutional ethical committee and informed consent was obtained from all participants included.

MAFLD was defined by evidence of hepatic steatosis on ultrasound or magnetic resonance imaging–based proton density fat fraction (MRI-PDFF) and coexistence of overweight/obesity, presence of T2DM, or evidence of metabolic dysregulation **(1)**.

Inclusion criteria: Metabolic associated fatty liver disease (MAFLD) criteria include adult patients with hepatic steatosis (steatosis detected by either imaging abdominal ultrasound, blood biomarkers/scores or by liver histology) with the presence one of the three criteria, overweight or obesity, type 2 diabetes mellitus or evidence of metabolic abnormalities such as an increased waist circumference and an abnormal lipid or glycemic profile. Lean patients (lean/normal weight defined as BMI<25kg/m^2 in Caucasian or BMI<23kg/m^2 in Asian) (1). Overweight / obese patients: (weight defined as BMI>25kg/m^2 in Caucasian or BMI>23kg/m^2 in Asian) (1).

Exclusion criteria were age less than 18 years, Patients with significant alcohol consumption (more than or equal to 30 g of alcohol per day for men and 20 g of alcohol per day for women), Hepatitis C positive patients, Hepatitis B positive patients, Patients with symptoms and signs suggesting other liver diseases: e.g. (Haemochromatosis, Wilson's disease, Alpha-one anti-trypsin deficiency, Autoimmune hepatitis), Patients with liver

cirrhosis and medicines known to be steatogenic (amiodarone, valproic acid, antiretroviral drugs, methotrexate, and tetracyclines), or medicines that are used for management of NAFLD (Vitamin E, metformin, and thiazolidinediones).

All patients were divided into 2 equal groups: Group I: included 45 lean MAFLD patients and Group II: included 45 obese MAFLD patients.

All patients were subjected to full history taking including age, sex, body mass index (BMI), occupation, residence and associated disease and history of drugs known to be steatogenic. Clinical examination: The weight and height of the participants was measured with a calibrated scale. Body mass index (BMI) was computed as body weight/ (height^2) (kg/M^2), blood pressure, waist circumference, General, systemic and local examination. Laboratory investigation including complete blood picture (C.B.C), Hemoglobin (g/dL), White blood cells (x10^3/□l), Platelets (x10^3/□l) and Fasting blood sugar and 2 hours post-prandial (mg/dL) and HbA1c (%). Markers of liver injury including serum alanine aminotransferase (ALT) (IU/L), serum aspartate aminotransferase (AST) (IU/L) and serum alkaline phosphatase (ALP) (IU/L), were done Liver function tests including serum total and direct bilirubin (mg/dl), Serum albumin (g/dl), Prothrombin time and the International Normalized Ratio (PT, INR). Serum creatinine (mg/dl). Viral markers including HBsAg by using 3rd generation enzyme linked immunosorbent assay technique (ELISA), Anti-HCV-Ab by using 3rd generation enzyme linked immunosorbent assay technique (ELISA).

Lipid profile including total cholesterol (TC) (mg/dl), triglycerides (TG) (mg/dl), high density lipoproteins (HDL) (mg/dl) and low-density lipoprotein (LDL) (mg/dl). Serum uric acid (mg/dl).

Imaging:

- Fibroscan with CAP (Controlled Attenuation Parameter) for assessment of hepatic steatosis: Transient Elastography using FibroScan® was performed by an experienced hepatologist using an XL probe, in patients who fasted for at least 6 hours prior to examination, in the supine position, with the right arm in full abduction, on the midaxillary line with the probe tip placed in the 9thto 11th intercostal space with a minimum of 10 measurements (6) . Liver stiffness (LS) values were regarded as valid if the following criteria were met: Number of valid measurements at least 10. A success rate above 60%. An interquartile range (IQR, reflecting the variability of measurements) less than 30% of the median LS measurements (M) value (IQR/M ≥30%) (6). The XL probe was used in this study due to the presence of morbidly obese patients. The measurement depth was between 35 and 75 mm.
- Controlled attenuation parameter (CAP) was also obtained to quantify degree of steatosis according to the manufacturer's instructions, in addition to previous studies, the stages of fibrosis (F0: 1–6, F1: 6.1–7, F2: 7–9, F3: 9.1–10.3, and F4: ≥ 10.4) were defined in kPa19, 20. Moreover, steatosis stages (S0: < 215, S1: 216–252, S2: 253–296, S3: > 296) were defined in dB/m (7).

Statistical analysis:

Statistical analysis was done by SPSS v26 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing

unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the Chi-square test. Pearson correlation was done to estimate the degree of correlation between two quantitative variables. Evaluation of Diagnostic Performance was performed using diagnostic sensitivity, specificity, PPV and NPV. Receiver Operating Characteristic curve (ROC-curve) analysis: The overall diagnostic performance of each test was assessed by ROC curve analysis, a curve that extends from the lower left corner to the upper left corner then to the upper right corner is considered a perfect test. The area under the curve (AUC) evaluates the overall test performance (where the area under the curve >50% denotes acceptable performance and area about 100% is the best performance for the test). A two tailed P value < 0.05 was considered statistically significant.

Research ethics committee: MD.10.3.2021

Results

Age, sex waist circumference, smoking and medication were insignificantly different between both groups. BMI was significantly lower in group I compared to group II (P **value** <0.001). Diabetes and Hypertension were insignificantly different between both groups. AST, total bilirubin and direct bilirubin were significantly lower in group I compared to group II (P value =0.001, 0.004 and 0.001 respectively) and TG was significantly higher in group I compared to group II (P value = 0.029). ALT, albumin, Cholesterol, LDL and HDL were insignificantly different between both groups. Fasting blood glucose, 2H post-prandial blood glucose, HbA1C and Serum uric acid were insignificantly different between both groups. Serum creatinine was significantly lower in group I compared to group II (P value =0.001). (Table 1)

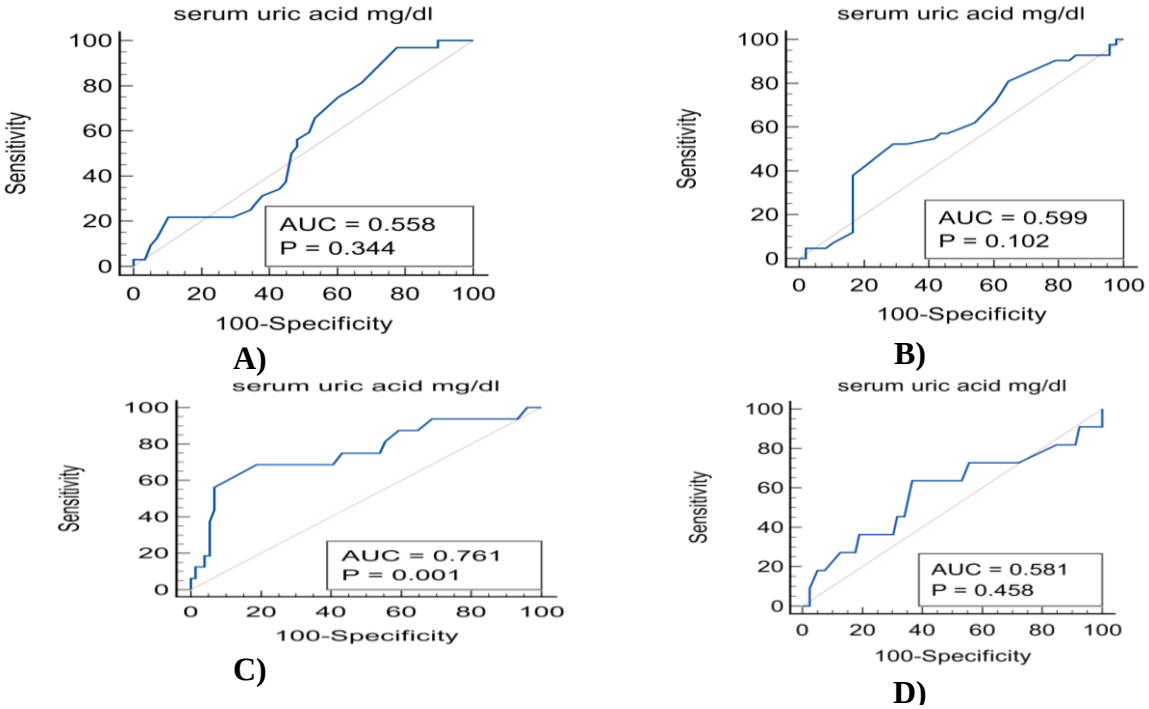

Figure 1: A) ROC curve of Serum uric acid in prediction of S1, B) ROC curve of Serum uric acid in prediction of S2, C) ROC curve of Serum uric acid in prediction of S3 and D) ROC curve of Serum uric acid in prediction of F2.

Table 1: Baseline characteristics and Clinical data of the studied groups.

		Group I (n=45)	Group II (n=45)	P value
Age (years)	**Mean ± SD**	48.27 ± 13.29	50.93 ± 12.96	0.338
	Range	23 - 67	22 - 75	
Sex	**Male**	23 (51.1%)	27 (60%)	0.528
	Female	22 (48.9%)	18 (40%)	
BMI (Kg/m^2)	**Mean ± SD**	48.27±13.29	50.93±12.96	**<0.001***
	Range	23-67	22-75	
Waist circumference (cm)	**Mean ± SD**	99.2±11.93	103.47±17.76	0.184
	Range	10-130	81-125	
Smoking		14 (31.1%)	13 (28.9%)	1
Medications		32 (71.1%)	36 (80%)	0.462
Hb (g/dL)		12.78±1.8	13.02±1.48	0.483
		8-16	10-16	
PLT (*10^{3}cells/µL)		330.71±113.96	330.29±76.26	0.984
		123-600	200-600	
WBCs (*10^{3}cells/µL)		6.16±2.24	6.04±2.33	0.818
		2-11	2-11	
ALT (U/L)		330.71 ± 113.96	330.29 ± 76.26	0.984
		123-600	200-600	
AST (U/L)		34.78 ± 18.26	49.62 ± 23.77	**0.001***
		12-87	15-123	
Total bilirubin (mg/dL)		0.63 ± 0.23	0.8 ± 0.32	**0.004***
		0.3-1.5	0.3-2	
Direct bilirubin (mg/dL)		0.23 ± 0.14	0.38 ± 0.26	**0.001***
		0.1-0.9	0.1-1.5	
Albumin (g/dL)		3.79 ± 0.52	3.7 ± 0.6	0.455
		2.7-4.8	2.2-5.4	
Cholesterol (mg/dL)		186.58 ± 54.44	200.31 ± 51.88	0.224
		130-400	130-400	
TG (mg/dL)		194.93 ± 119.68	154.29 ± 28.45	**0.029***
		109-700	100-222	
LDL (mg/dL)		114 ± 48.12	132.91 ± 46	0.060
		65 -310	80 - 318	
HDL (mg/dL)		56.27±7.46	55.18±8.29	0.514
		40-70	40-70	
Serum uric acid (mg/dl)		5.42±1.43	5.64±1.75	0.528
		3.7-11	3.1-11.1	

BMI: body mass index, HBV: hepatitis B virus, HCV: hepatitis C virus, CBC: complete blood count, Hb: Hemoglobin, PLT: platelet count, RBCs: red blood cells, WBCs: white blood cells, ALT: alanine aminotransferase, AST: aspartate aminotransferase, TG: triglycerides, LDL: low-density lipoprotein, HDL: high-density lipoprotein, *: statistically significant as P value <0.005.

There was a significant positive correlation between serum uric acid and cholesterol (r=0.300, P=0.004), LDL (r=0.301, P=0.00) and CAP score (r=0.289, P=0.006), and there was an insignificant correlation between serum uric acid and other different parameters. (Table 2)

Serum uric acid can significantly predict the incidence of S3 with AUC 0.761 and P value = 0.001, at cut off value >4.9 mg/dL, 87.5 % sensitivity, 40.54% specificity, 24.1 PPV and 93.8 NPV. Serum uric acid is an insignificant predictor of S1, S2 and F2. (Table 3 and Figure 1)

Table 2: Correlation between serum uric acid and different parameters.

	r	p
Age (years)	0.113	0.287
Fasting blood glucose (mg/dl)	0.073	0.493
2H post-prandial blood glucose (mg/dl)	0.069	0.518
HbA1C (%)	0.096	0.369
BMI (Kg/m^2)	0.150	0.159
Waist circumference (cm)	0.203	0.055
Hb (g/dL)	0.023	0.826
WBCs (*10^3cells/µL)	0.047	0.659
Platelets (*10^3cells/µL)	0.060	0.571
ALT (U/L)	0.206	0.052
AST (U/L)	0.127	0.231
Total bilirubin (mg/dL)	0.091	0.400
Direct bilirubin (mg/dL)	0.043	0.686
Albumin (g/dL)	-0.045	0.675
Serum creatinine (mg/dl)	0.056	0.603
Cholesterol (mg/dL)	0.300	**0.004***
TG (mg/dL)	0.131	0.218
LDL (mg/dL)	0.301	**0.004***
HDL (mg/dL)	0.038	0.719
CAP score	0.289	**0.006***
Liver stiffness measurements	0.009	0.936

Hb: Hemoglobin, PLT: platelet count, RBCs: red blood cells, WBCs: white blood cells, ALT: alanine aminotransferase, AST: aspartate aminotransferase, TG: triglycerides, LDL: low-density lipoprotein, HDL: high-density lipoprotein, CAP: controlled attenuation parameter, S: steatosis, *: statistically significant as P value <0.005.

Table 3: Diagnostic performance of serum uric acid in prediction of S1, S2, S3 and F2.

	AUC	**P value**	**Cutoff**	**Sensitivity**	**Specificity**	**PPV**	**NPV**
S1	0.558	0.344	≤5.7	75	39.66	40.7	74.2
S2	0.599	0.102	≤5.9	80.95	35.42	52.3	68
S3	0.761	0.001*	>4.9	87.5	40.54	24.1	93.8
F2	0.581	0.458	≤5.6	72.73	44.3	15.4	92.1

AUC: Area under the curve, PPV: positive predictive value, NPV: negative predictive value, S: steatosis, F: fibro scan, *: statistically significant as P value <0.05.

Discussion

Fatty liver disease (FLD) is prevalent worldwide with an estimated prevalence of 25%. As a result of the insidious onset and prolonged course, the patient pool is continuously increasing. Based on the history of alcohol intake, it is currently artificially classified into two common forms: alcoholic liver disease (ALD) and non-alcoholic fatter liver disease (NAFLD) (8).

Interestingly, a study performed a multivariate logistic regression model to identify the influencing factors of MAFLD, and the goodness of fit test showed that the regression model had statistical significance ($\chi^2 = 8985.31$, $p < 0.001$). The analysis revealed that BMI, waist circumference, LYMPH%, HGB

level, PLT level, ALT level, TG level, FPG level, and SUA level were independently and positively correlated with the presence of MAFLD (all $p < 0.05$); in contrary, active physical activity and HDL-C level were independently and negatively correlated with the presence of MAFLD (all $p < 0.05$) (9).

In contrast, a study reported that by performing univariate logistic regression analysis that HOMA-IR, TG, uric acid, and ferritin levels were associated with liver steatosis. In the multivariate logistic regression analysis, only high TG levels were significantly associated with liver steatosis after adjusting for confounding factors. This may be because Chinese people have their own lifestyle and genetic characteristics, which are different from Middle East population (10).

Came in line with our results, a study found that when performing a similar analysis in MAFLD quantified with MRI-PDFF, for moderate-to-severe steatosis (21.7% ≥LFC ≥ 16.3%), the cutoff value of SUA (≥438.5 μmol/L in males and ≥403.5 μmol/L in females) and the corresponding AUC (0.676 in males, 95% CI 0.635–0.718; and 0.601 in females, 95% CI 95% CI 0.523–0.679, both p < 0.01) were similar. For predicting severe steatosis (LFC ≥ 21.7%), the cutoff values of SUA increased to ≥467.0 μmol/L in males and ≥431.5 μmol/L in females, with AUCs of 0.672 (95% CI 0.620–0.724, p < 0.01) and 0.577 (95% CI 0.474–0.680, p = 0.11) in males and females, respectively (11).

In agreement with our study, a trial found that in MAFLD patients, similar results were observed for most aspects. They reported an association between SUA levels and moderate-to-severe steatosis (liver fat content ≥ 16.3%), and the OR increased from 2.20 (95% CI 1.29–3.77) to 2.28 (95% CI 1.93–558). However, these associations were not found for females. Also, the association between SUA levels and severe steatosis measured with MRI-PDFF persisted (11).

In harmony with the current work, a trial found that the Univariate logistic regression analysis showed that age and high uric acid level were associated with advanced liver fibrosis. In the multivariate logistic regression analysis, only high uric acid levels were a statistically significant predictor of advanced liver fibrosis (10).

In the same line with the current study, a cross-sectional was carried out in a Chinese population to explore the association between steatosis severity and SUA levels in MAFLD. Steatosis was estimated with ultrasound and magnetic resonance imaging–based proton density fat fraction (MRI-PDFF). Moreover, they examined whether SUA levels can be used as a diagnostic marker in lean/normal-weight MAFLD. All patients were divided into four groups according to their baseline SUA levels and sex. Of the 3537 ultrasound-diagnosed and 1017 MRI-PDFF-diagnosed MAFLD patients included, the prevalence of severe steatosis determined with ultrasound or MRI-PDFF increased across the serum SUA quartiles. They reported similar results regarding the AST, and TG in the MAFLD Defined by MRI-PDFF group (11).

This was consistent with a recent study where they found MAFLD patients with fibrosis had significant increased cholesterol, triglyceride, and serum uric acid (12).

Controlled attenuation parameter (CAP) score was insignificantly different between the studied groups. The Liver stiffness measurement (LSM) by fibro scan was

insignificantly different between the studied groups. Our results are compatible with a cross-sectional, community-based survey with multistage stratified cluster sampling to estimate the prevalence and risk factors of MAFLD among Beijing adults aged ≥25 years old. Demographic, transient elastography (TE), biochemical and blood examination information was collected in all the subjects in this study. Compared with subjects without MAFLD, the MAFLD patients drank more alcohol daily and had higher value of LSM and UAP ($p > 0.05$). Higher LSM and UAP value were observed in participants with lean MAFLD than in the non-MAFLD group ($p < 0.001$) (13).

Also, our study revealed that there was a significant relationship between CAP score and smoking (P value =0.034), whereas there was an insignificant relationship between CAP score and sex, DM, HTN and medications. There was a significant positive correlation between CAP score and Albumin (r=0.222, P=0.036), cholesterol (r=0.333, P=0.001) and serum uric acid (r=0.289, P=0.006) and there was an insignificant correlation between CAP score and other different parameters. There also was a significant positive correlation between serum uric acid and cholesterol (r=0.300, P=0.004), LDL (r=0.301, P=0.00) and CAP score (r=0.289, P=0.006), and there was an insignificant correlation between CAP score and other different parameters.

Also, a recent cross-sectional study was performed using data from a community screening examination for metabolic syndrome. A total of 182 lean subjects were included and were divided into lean MAFLD and lean healthy groups. They reported that lean MAFLD subjects were older and had a higher percentage of diabetes, metabolic syndrome, or hyperuricemia than lean healthy subjects. Lean MAFLD subjects had more metabolic abnormalities (waist circumference, blood pressure, TG, HDL, fasting glucose, HbA1c, IR, HS-CRP, and fatty liver index), liver enzymes, inflammatory markers, and higher noninvasive hepatic fibrosis scores ($p < 0.01$) (10).

Regarding the current work, serum uric acid can significantly predict the incidence of S3 with AUC 0.761 and P value = 0.001, at cut off value >4.9 mg/dL, 87.5 % sensitivity, 40.54% specificity, 24.1 PPV and 93.8 NPV. Serum uric acid is an insignificant predictor of S1, S2 and F2.

To the best of our knowledge, no study has been performed so far to investigate the association of the level of SUA with MAFLD.

Conclusion

Our study supports the rationale for Serum uric acid (SUA) being established as another risk factor for metabolic dysfunctions in lean/normal-weight and obese MAFLD. It is considered as an inexpensive noninvasive biomarker for evaluating advanced liver fibrosis and combining it with other scoring systems may help to improve its predictive power. We also suggest that SUA could be used as a clue to the severity of MAFLD. There is evidence to suggest that there may be an association between MAFLD and SUA levels as in the current study did. Some studies have shown that MAFLD is positively associated with SUA levels, while others have found no significant association. Therefore, further studies are needed to explore the association.

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To cite this article: Badawy A. Abdulaziz , Magdy A.Gad , Ibrahim M. Ghadour , Walid A. Abdellhalim , Ahmed S. Elgazar. Serum Uric Acid in Metabolic Associated Fatty Liver Disease in Obese and Lean Patients. BMFJ 2023;40(2): 411-419.