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Investigation of hemogram and biochemistry parameters in non-alcoholic fatty liver disease

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is primarily characterized by the buildup of triglycerides in the hepatocytes. The present study sought to compare the hemogram and biochemistry parameters of patients with different grades of hepatic steatosis (HS) detected in abdominal ultrasonography (USG) examinations with the healthy control group. Patients who sought treatment at the internal medicine outpatient clinic for various causes and underwent abdominal USG were included in the study. According to USG results, participants were subdivided into four groups based on their HS grade 0-1-2-3. The mean age and gender distributions of the groups were not statistically different (p=0.306 and p=0.457, respectively). There was no statistical difference between the groups in terms of smoking status (p=0.344). It was determined that the prevalence of obesity, DM and HT increased as the HS grade increased (p<0.001, p=0.015, p<0.001). Mean platelet volume (MPV), atherogenic index of plasma (AIP), and fasting triglyceride levels were significantly different between the groups (p=0.012, p=0.008, and p=0.003, accordingly). Uric acid, uric acid albumin ratio (UAR), and AIP values and HS grade were positively and significantly correlated (p=0.036 r=0.276, p=0.024 r=0.294, and p=0.013 r=0.308, accordingly). When we considered HS grade as the dependent variable, the UAR value predicted HS grade positively and significantly according to linear regression analysis (p=0.008, Beta=0.387). According to the ROC curve analysis, UAR was found to be the most significant in the diagnosis of NAFLD (sensitivity 67%, specificity 64%, cut-off value:1.05). From a practical point of view, healthcare professionals need to be alerted that patients with co-existing hyperuricemia and elevated AIP are at greater risk of progressing to hepatic steatosis and, as a result, cardiovascular diseases. Additionally, our study confirms the need for more extensive longitudinal studies to understand the processes contributing to the relationship between UAR, AIP, and NAFLD. UAR value can be used as an easily accessible marker that can be used to show fatty liver. Future studies can examine the influence of reducing uric acid levels on the fatty liver.

Keywords: Non-alcoholic fatty liver disease, hepatic steatosis, uric acid, albumin, atherogenic index of plasma

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a health issue that has increased in importance and frequency with increased living standards in recent years. NAFLD is primarily characterized by the buildup of triglycerides in the hepatocytes. Although fatty liver usually follows a mild course, it can sometimes progress and lead to fibrosis and cirrhosis of the liver. While the pathological appearance is strikingly similar to liver damage caused by alcohol consumption, patients with NAFLD do not have heavy

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alcohol consumption [1].

The true prevalence of NAFLD is unknown, but it is thought to be the most prevalent liver disease in both industrialized and developing nations. Its incidence in various societies in the world varies between 10–24%. NAFLD is a condition characterized by a low-grade inflammatory reaction in the fatty tissue of the liver parenchyma, together with pathological fat accumulation and insulin resistance. Therefore, insulin resistance is closely related to oxidative stress and endothelial dysfunction markers [2].

It is thought that phosphorylation of fructose used as a sweetener by fructokinase induces triglyceride synthesis by triggering purine degradation and, in this way, causes the development of NAFLD. The entry of AMP, produced through the activation of AMP deaminase, into the purine degradation pathway leads to the formation of uric acid mitochondrial oxidants. These oxidants disrupt the Krebs cycle and lead to citrate accumulation and fatty acid synthesis by activating the ATP citrate lyase enzyme [3].

The present study sought to compare the hemogram and biochemistry parameters of patients with different grades of hepatic steatosis (HS) detected in abdominal ultrasonography (USG) examinations with the healthy control group.

Materials and Methods

Study Design

The present study is a retrospective study. In this study, participants applied to the hospital between 01.03.2022 and 01.09.2022. The method of this study was confirmed by the university's ethics committee (IRB Number:2022/7-47). The study was undertaken in accordance with the Declaration of Helsinki. Patients who sought treatment at the internal medicine outpatient clinic for various causes and underwent abdominal USG were included in the study. The control group was comprised of people who had abdominal USG for abdominal pain, weight loss, anorexia, and indigestion but without hepatosteatosis or any pathology. Those with regular alcohol consumption were not included in the study. According to abdominal USG results, participants were subdivided into four groups based on their HS grade 0-1-2-3. The body-mass index (BMI) of the participants was calculated by dividing their weight by the square of their height (kg/m²). The smoking status of the participants, comorbid diseases such as obesity, diabetes mellitus (DM) and hypertension (HT) were recorded. Groups were selected similar in age and gender. Care was taken to ensure that the number of patients in the groups was close to each other.

Laboratory Examination

Neutrophil lymphocyte ratio (NLR), platelet lymphocyte ratio (PLR), monocyte lymphocyte ratio (MLR), uric acid albumin ratio (UAR), monocyte high-density lipoprotein cholesterol (HDL-C) ratio (MHR), atherogenic index of plasma (AIP) were analyzed. AIP was calculated by taking the logarithm of the triglyceride ratio to HDL-C [4].

Ultrasonographic Examination

Radiologist with 5-year experience performed abdominal USG. According to the literature, normal liver echo on USG is considered HS grade 0. A diffuse and mild increase in liver parenchyma echo on USG is referred to as HS grade 1. HS grade 2 is defined as the inability to see the portal vein and diaphragm margins clearly as well as a moderate increase in liver parenchyma echo in USG. In USG, HS grade 3 is defined as only limited or no detection of the portal vein, diaphragm, and the posterior side of the right lobe of the liver and the presence of liver parenchyma as fine echoes [5].

Statistical Analysis

All analyzes were conducted using SPSS Mac Version 26.0. Categorical data were shown as numbers and percentages, and continuous data were reported as mean ± standard deviation or median (minimum: maximum). The normality of continuous data was verified with the Kolmogorov-Smirnov test. A one-way ANOVA test was employed for multiple comparisons of normally distributed data, and a student-t-test was used for pairwise comparisons. The Tukey test was used for post hoc analysis in a one-way ANOVA. The Kruskal-Wallis test was employed for multiple comparisons of non-normally distributed data, and the Mann-Whitney U test was applied for pairwise comparisons. Receiver operating characteristic (ROC) analysis was used for determination of sensitivity and specificity of lab parameters on diagnosing NAFLD. $p<0.05$ was evaluated as statistically significant.

Results

Comparison of sociodemographic features and electrocardiographic parameters of patients with hepatic steatosis and healthy controls was shown in Table 1. Mean age and gender distribution of the groups: HS grade 0 (42.45±7.95) (F:12, M:8), grade 1 (44.80±8.30) (F:10, M:10), grade 2 (47.35±5.74) (F:14, M:6), and grade 3 (43.86±10.54) (F:10, M:10). The mean age and gender distributions of the groups were not significantly different ($p=0.306$ and $p=0.457$, respectively). There was a significant difference between the groups in terms of BMI ($p=0.022$). There was no statistical difference between the groups in terms of smoking status ($p=0.344$). It was determined that the prevalence of obesity, DM and HT increased as the HS grade increased ($p<0.001$, $p=0.015$, $p<0.001$). Comparison of laboratory and inflamatory parameters of patients with hepatic steatosis and healthy controls was shown in Table 2. According to the Kruskal Wallis test, MPV, AIP, and fasting triglyceride levels were significantly different between the groups ($p=0.012$, $p=0.008$, and $p=0.003$, respectively). NLR, PLR, MLR, AIP, and UAR did not differ between the groups ($p=0.515$, $p=0.372$, $p=0.287$, $p=0.990$, $p=0.116$). Correlation analyzes of hepatic steatosis grade with laboratory parameters was shown in Table 3. According to Pearson correlation analysis, uric acid, UAR, and AIP values and HS grade were positively and significantly correlated ($p=0.036$ $r=0.276$, $p=0.024$ $r=0.294$, and $p=0.013$

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Table 1. Comparison of Sociodemographic Features and Electrocardiographic Parameters of Patients with Hepatic Steatosis and Healthy Controls

	HC (n=20) M±SD or n (%)	HS G1 (n=20) M±SD or n (%)	HS G2 (n=20) M±SD or n (%)	HS G3 (n=21) M±SD or n (%)	P
Age	42.45±7.97	44.80±8.30	47.35±5.74	43.86±10.54	0.306[1]
Gender					0.457[2]
Female	12 (60)	10 (50)	14 (70)	10 (47.6)	
Male	8 (40)	10 (50)	6 (30)	11 (52.4)	
BMI (kg/m²)	26.24±4.66[a]	28.7±5.62[b]	29.8±5.44[b]	33.95±6.2[c]	0.022[1]
Smoking	6 (30)	7 (35)	5 (20)	6 (28.6)	0.344[2]
Obesity	1 (5)	3 (15)	7 (35)	12 (57)	<0.001[2]
DM	1 (5)	2 (10)	3 (15)	6 (28.6)	0.015[2]
HT	1 (5)	2 (10)	6 (30)	10 (47.7)	<0.001[2]

HC, healthy controls; HS, hepatic steatosis; G, grade; BMI, body-mass index; DM, diabetes mellitus; HT, hypertension [1]One-way ANOVA test was used. [2]Chi-square test was used. p<0.05 was accepted as statistically significant

Table 2. Comparison of Laboratory and Inflamatory Parameters of Patients with Hepatic Steatosis and Healthy Controls

	HC (n=20) M±SD or n (%)	HS G1 (n=20) M±SD or n (%)	HS G2 (n=20) M±SD or n (%)	HS G3 (n=21) M±SD or n (%)	P
Albumin, mg/dl	4.10 (2.60:4.90)	4.10 (3.40:4.50)	4.10 (3.70:4.70)	4.10 (3.50:4.60)	0.858[2]
Uric acid, mg/dl	4.09±1.33	4.44±0.89	4.71±1.24	5.30±1.05	0.054[1]
Glucose, mg/dl	97 (74:169)	98 (78:205)	106 (71:204)	107 (84:246)	0.125[2]
Neutrophil, 10^6/µL	5.07±1.32	4.43±1.47	4.27±1.49	4.87±2.66	0.496[1]
Lymphocyte, 10^3/µL	2.38±0.59	2.17±0.55	2.55±0.60	2.66±0.78	0.093[1]
Platelet, 10^3/µL	259.42±82.37	256.90±63.35	270.30±64.62	258.33±64.22	0.924[1]
Monocyte, 10^3/µL	0.57 (0.30:1.13)	0.52 (0.36:0.96)	0.51 (0.27:1.20)	0.56 (0.30:1.04)	0.571[2]
MPV, fL	8.26[a] (6.34:10.40)	8.92[b] (7.41:11.60)	8.51[a,b] (7.74:12.20)	8.26a (7.07:10.30)	0.012[2]
Fasting Triglyceride, mg/dL	94[a] (30:321)	149b,c (81:632)	132[b] (70:335)	178[c] (89:293)	0.003[2]
Total-C, mg/dL	173 (122:336)	182 (138:225)	197.5 (143:265)	178 (127:253)	0.100[2]
LDL-C, mg/dL	100.69±39.68	108.56±22.49	123.61±34.70	98.82±32.08	0.117[1]
HDL-C, mg/dL	47 (28:99)	43 (26:59)	41.5 (30:61)	43 (35:59)	0.737[2]
NLR	2.24±0.77	2.07±0.63	1.86±1.20	1.88±0.93	0.515[1]
PLR	115.35±53.54	123.35±39.59	109.76±26.94	102.14±33.46	0.372[1]
MLR	0.25 (0.15:0.51)	0.25 (0.15:0.36)	0.19 (0.10:0.44)	0.19 (0.10:0.48)	0.287[2]
MHR	0.01 (0.01:0.02)	0.01 (0.01:0.03)	0.01 (0.01:0.03)	0.01 (0.01:0.02)	0.990[2]
AIP	0.26±0.35[a]	0.59±0.30[b]	0.49±0.24[a,b]	0.60±0.19[b]	0.005[1]
UAR	1.04±0.40	1.10±0.23	1.13±0.31	1.32±0.26	0.116[1]

HC, healthy controls; HS, hepatic steatosis; G, grade; MPV, mean platelet volume; Total-C, total cholesterol; LDL-C, low-density cholesterol; HDL-C, high-density cholesterol; NLR, neutrophil lymphocyte ratio; PLR, platelet lymphocyte ratio; MLR, monocyte lymphocyte ratio; MHR, monocyte HDL-C ratio; AIP, atherogenic index of plasma; UAR, uric acid albumin ratio. [1]One-way ANOVA test was used. [2]Kruskall-Wallis test was used. Student's t test was used for pairwise comparisons in normally distributed data, and Mann-Whitney U test was used in non-normally distributed data. p <0.05 was accepted as statistically significant.

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Table 3. Correlation Analyzes of Hepatic Steatosis Grade with Laboratory Parameters

	Hepatic Steatosis Grade
Albumin	r=0.82
	p=0.476
Uric acid	r=0.276
	p=0.036
UAR	r=0.294
	p=0.024
AIP	r=0.308
	p=0.013
Fasting Triglyceride	r=0.185
	p=0.141
MPV	r=-0.027
	p=0.813

UAR, uric acid albumin ratio; AIP, atherogenic index of plasma; MPV, mean platelet volume. Pearson corelation analyzes was used. p<0.05 was accepted as statically significance

Table 4. Linear Regression Analyzes of Hepatic Steatosis Grade

	B	Std. Error	Beta	t	P	95 % CI Lower	Upper
Constant	0.896	1.211		0.740	0.463	-1.536	3.327
AIP	0.170	0.562	0.046	0.302	0.764	-0.959	1.298
UAR	0.387	0.478	0.376	2.624	0.008	0.294	2.213
MPV	0.027	0.125	0.031	0.219	0.828	-0.223	0.278

AIP, atherogenic index of plasma; UAR, uric acid albumin ratio; MPV, mean platelet volume. Linear regression analyses was used. p<0.05 was accepted as statically significance. (F(3,50)=6.133, Adjusted R2=.432 and p<0.001)

r=0.308, accordingly). Linear regression analyzes of hepatic steatosis grade was shown in Table 4. When we considered HS grade as the dependent variable, the UAR value predicted HS grade positively and significantly according to linear regression analysis (p=0.008, Beta=0.387). ROC curve analyzes of lab parameters was shown in Table 5 and Figure 1. According to the ROC curve analysis, UAR was found to be the most significant in the diagnosis of NAFLD (sensitivity 67%, specificity 64%, cut-off value:1.05).

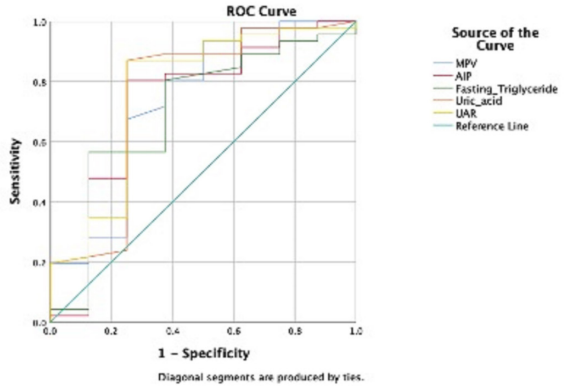

Figure 1. ROC Curve of Lab Parameters for Diagnosing NAFLD

MPV, mean platelet volume; AIP, atherogenic index of plasma; UAR, uric acid albumin ratio

Table 5. ROC Curve Analyzes of Lab Parameters

Variable	Area	p
MPV	0.736	0.034
AIP	0.731	0.038
Fasting Triglyceride	0.701	0.072
Uric Acid	0.755	0.022
UAR	0.768	0.016

Discussion

Serum albumin is the most prominent and abundant protein in the blood and plays a number of essential metabolic roles. The elevated inflammatory process is accompanied by decreased production and increased degradation of serum albumin. Low serum albumin levels may boost blood viscosity and lead to endothelial dysfunction [6]. Moreover, serum albumin is an essential inhibitor of platelet activity and aggregation and an effective modulator of platelet-driven coronary stenosis [7].

Uric acid is produced during purine metabolism, and its high levels increase the risk of atherosclerosis and determine the prognosis of coronary artery disease. Uric acid is one of the

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mediators of vascular disease, endothelial dysfunction, and inflammation. It has been shown that hyperuricemia is associated with coronary atherosclerosis with a high plaque-loaded risk [8]. Epidemiological evidence has shown that hyperuricemia and cardiovascular diseases are associated. It has been established that increased uric acid levels are an independent predictor of cardiovascular disease and cardiovascular mortality. However, the causal mechanisms linking high uric acid levels to cardiovascular disease are still unclear. Uric acid tends to crystallize and generate monosodium urate crystals in several tissues and initiates regional immune activity. The atherosclerotic plaque comprises substantial concentrations of uric acid, and elevated levels of uric acid promote thrombus formation. In addition, uric acid can lead to oxidative stress and cause inflammation, vasoconstriction, and endothelial dysfunction [9].

A number of investigations have concluded that low serum albumin levels are connected to the existence of coronary artery disease and an elevated risk of overall mortality. There is also considerable proof that low serum albumin concentrations can lead to the formation and worsening of atherosclerosis. In coronary artery disease, hypoalbuminemia plays a part in the pathogenesis by resulting in a decrease in anti-inflammatory, antioxidant, and anti-thrombotic activities [10].

However, uric acid and serum albumin could not be included in any risk profile. First, uric acid levels are affected by renal excretion, consumption of various nutrients, and uric acid cell metabolism. In addition, abnormally high uric acid levels are usually related to gout, insulin resistance, diabetes, metabolic syndrome, hypertension, and chronic kidney disease. Serum albumin synthesis is promoted by: amino acid uptake, insulin, and low colloid osmotic pressure. The components that reduce albumin production are increased colloid osmotic pressure, inflammation, nutritional deficiency, diabetes, liver disease, and infections [11].

NAFLD has emerged as the foremost liver disease globally, caused by lipid metabolism disorders and insulin resistance. AIP, an estimated lipid measurement, is a new indicator for insulin resistance [12]. However, the association between plasma atherogenic index and NAFLD was uncertain, and scientific literature examining the relationship between AIP and NAFLD were scarce. The first study about this matter investigated the relationship between AIP and NAFLD in 538 obese participants. They showed that the risk of NAFLD increased 4.37 times in the high plasma atherogenic index group [13]. In 2014, Akbaş et al. found AIP to be higher in those with high uric acid levels, and they found that AIP is an independent predictor of hyperuricemia in diabetic patients [14]. Xie et al. investigated the association between AIP and NAFLD and showed that the risk of NAFLD increased as AIP increased, especially in women and young people [15]. In 2020, Dong et al. investigated the diagnostic value of AIP in demonstrating NAFLD in normal weight people and confirmed that AIP is a distinct risk marker in demonstrating NAFLD [16].

Many researchers have reported that elevated serum uric acid concentration is potential risk factor for cardiovascular diseases. The significant relationship between serum uric acid and dyslipidemia as aspects of metabolic syndrome has been examined in various studies. Many reasons have been suggested to elucidate the relation between hyperuricemia and cardiovascular disease risk factors. Reduced renal clearance of uric acid or higher proximal tubular reabsorption resulting from insulin resistance, elevated leptin concentrations, and excessive fructose intake commonly connected with obesity have been suggested to be potential reasons for this heightened cardiovascular disease risk [17].

It has been reported in the literature that HDL-C is decreased in the population with high uric acid. Some studies have suggested that hypertriglyceridemia reduces the renal excretion of uric acid. In addition, the heritability of high uric acid and atherosclerosis in twins supports that the underlying pathogenic mechanisms are related [18]. Previous studies have shown that uric acid induces lipid synthesis in hepatocytes. It is emphasized that uric acid may predict the risk of hypertension, type 2 diabetes mellitus, and cardiovascular disease [19].

The present study has a number of limitations. Firstly, this study is inconclusive due to its retrospective methodology. The retrospective data acquisition process may miss certain variables. Second, because it covers only one hospital, it may not reflect the entire population. Lastly, our study's retrospective design prevents us from detecting causality.

Conclusions

From a practical point of view, health care professionals need to be alert that patients with co-existing hyperuricemia and elevated AIP are at elevated risk of progressing to hepatic steatosis and, as a result, cardiovascular diseases. Additionally, our study confirms the need for more extensive longitudinal studies to understand the processes contributing to the relationship between UAR, AIP, and NAFLD. UAR value can be used as an easily accessible marker that can be used to show fatty liver. In future studies, the influence of reducing uric acid levels on the fatty liver can be examined.

Conflict of interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical approval
The method of this study was confirmed by the Adıyaman University Non-Interventional Clinical Researches Ethics Committee (IRB Number:2022/7-47).

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