Original Article

Does platelet to lymphocyte ratio predict the ultrasound stage in hepatosteatosis?

Acta Radiologica
2025, Vol. 66(6) 660–665

DOI: 10.1177/02841851251322480
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Abstract

Background: The prevalence of hepatosteatosis, or fatty liver disease, has been increasing globally in recent years largely due to increasing rates of obesity, diabetes, and metabolic syndrome.
Purpose: To examine the platelet to lymphocyte ratio (PLR) reflection on the hepatosteatosis stage.
Material and Methods: We evaluated healthy individuals who applied to the check-up department in our hospital. The platelet and lymphocyte counts from blood tests, along with upper abdominal ultrasound results obtained as part of routine diagnostic check-ups, results recorded retrospectively, between November 2022 and April 2024.
Results: A total 748 participants were included in the study. All participants were divided in three groups according to hepatosteatosis stages.The PLR levels were highest in the stage 1 hepatosteatosis group. There was statistical significance in PLR levels between stage 1 and 3 hepatosteatosis ($P = 0003$). In addition, PLR levels were higher in stage 2 than in stage 3, which was also statistically significant ($P = 0037$).
Conclusion: These results could help in early detection and monitoring of disease progression in patients with hepatoteatosis. Lower PLR values (<115.26) in advanced stages might prompt closer monitoring or more aggressive interventions to prevent progression to fibrosis.

Keywords
Hepatosteatosis, stage, platelet to lymphocyte ratio, ultrasound

Date received: 28 August 2024; accepted: 20 January 2025

Introduction

Hepatosteatosis, also known as fatty liver disease or non-alcoholic fatty liver disease (NAFLD), refers to a condition in which fat accumulates in liver cells with no alcoholic intake and where other diseases have been excluded. This fat accumulation can cause inflammation and damage to the liver, leading to decreased liver function over time (1). The prevalence of hepatosteatosis, or fatty liver disease, has been increasing globally over the past few decades, largely due to increasing rates of obesity, diabetes, and metabolic syndrome. NAFLD is the most common form of hepatosteatosis. It is estimated that approximately 25% of the global adult population has NAFLD. In certain regions, particularly in Western countries, the prevalence of NAFLD is notably higher, with estimates in the range of 25%–45% across different studies (2,3). A population-based study from the United States revealed that the risk of hepatocellular carcinoma (HCC) increases by 59% in patients diagnosed with NAFLD (4).

Platelet-derived factors play a significant role in modulating the immune response and influencing tumor progression. An increase in platelet count and the factors they release can contribute to cancer progression by suppressing the antitumor immune response and promoting tumor development and metastasis. Platelets can protect circulating tumor cells (CTCs) from being detected and eliminated by natural killer (NK) cells. By cloaking these CTCs, platelets reduce the effectiveness of NK cell-mediated cytotoxicity. Platelet-derived factors can also suppress the activity of cytotoxic T cells that are crucial for recognizing and destroying tumor cells. This immunosuppressive effect

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creates a tumor-favorable environment. Platelets are rich in VEGF, a potent pro-angiogenic factor. By enhancing vascularization, platelets help tumors acquire the necessary nutrients and oxygen, facilitating their growth and the spread of cancer to distant organs. Platelets play a key role in the metastatic cascade by shielding CTCs from immune surveillance, increasing their survival in the bloodstream, and facilitating their extravasation into distant tissues, ultimately driving metastatic progression (5). Lymphocytes are crucial for the immune response. A low lymphocyte count is often linked to immunosuppression and is commonly seen in patients with severe cancers, such as chronic hepatitis B virus (HBV) infection and HCC (6).

The platelet-to-lymphocyte ratio (PLR) is increasingly recognized as a useful, non-invasive biomarker for predicting the prognosis and treatment outcomes in liver cancer, particularly HCC, which is the most common type of liver cancer (7). Recent studies have found that the PLR is closely associated with disease severity in HBV and hepatitis C virus (HCV)-related liver diseases (8).

Chronic inflammation over an extended period contributes to disease progression and increases the likelihood of developing end-stage liver disease. Previous research has shown an association between IL-6 and C-reactive protein with disease advancement (9,10). Similarly, the FIB-4 index, a blood-based diagnostic tool for identifying underlying fibrosis in NASH, has been used for this purpose (11).

The aim of the present study was to critically evaluate the predictive value of PLR in accurately determining the radiological staging of hepatosteatosis, as identified through ultrasound imaging.

Material and Methods

This was a retrospective study and approval from the ethics committee was obtained (ATADEK 2024–8/324).

We retrospectively reviewed data from patients who underwent blood tests and upper abdominal ultrasound in our hospital's check-up department between November 2022 and April 2024. A medical check-up, also known as a health check-up or physical examination, is a routine assessment conducted by a healthcare professional to evaluate a person's overall health. It typically involves a medical history review, physical examination, laboratory blood tests (e.g. cholesterol, blood sugar), urine tests, and sometimes other screenings depending on age, sex, and medical history (e.g., mammograms, Pap smears, or prostate exams), and specialized screenings like X-rays, ultrasounds, or electrocardiograms (ECGs).

Patients with a history of HBV or HCV, those diagnosed with chronic liver disease, and individuals with a history of chronic alcohol consumption were excluded from the study.

A total of 748 participants were included in this retrospective cross-sectional study. The age, sex, weight, height, platelet count, upper abdominal ultrasound imaging information, co-morbidities, and fatty liver stage of these cases were recorded from the hospital data.

Abdominal ultrasound examinations for hepatosteatosis grading were performed after a fasting period of at least 8 h to ensure optimal imaging conditions, minimizing gastrointestinal content that could interfere with the ultrasound accuracy. The ultrasound examinations were conducted using a General Electric Logiq P10 device with a C1–6 convex probe, operating at an average frequency of 4 MHz. A radiology specialist with 22 years of experience in ultrasound imaging performed all assessments, ensuring consistency and reliability.

Liver steatosis grading was based on specific ultrasound features, including liver brightness (higher fat content increases echogenicity), the contrast between the liver and kidney (a more echogenic suggests a higher fat content), visibility of intrahepatic vessels (increased fat can obscure them), liver parenchyma homogeneity, and diaphragm appearance (significant steatosis may cause diaphragm flattening or elevation).

Steatosis is classified into three grades: mild (grade 1) = slight and diffuse increase in liver echogenicity with normal visualization of the diaphragm and portal vein wall; moderate (grade 2) = moderate increase in echogenicity with slightly impaired visualization of the portal vein wall and diaphragm; and severe (grade 3) = marked increase in liver echogenicity with poor or no visualization of the portal vein wall, diaphragm, and the posterior part of the right liver lobe (Image 1).

Blood samples were collected from peripheral veins after an 8-h fasting period and analyzed within 2 h of collection. Biochemical assessments, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, were conducted using an automated biochemical analyzer (Siemens Dimension). The platelet and lymphocyte count were detected from routine complete blood count (CBC) measured using a routine automated flow cytometer Sysmex XN20. The PLR was calculated by dividing the platelet count by the lymphocyte count in the blood sample. This ratio was then compared with the radiological staging of hepatosteatosis.

FIB-4 index was calculated using a $(age*AST)/(platelet\ count*\sqrt{ALT})$ formula. Scores were classified into low (<1.30), indeterminate (1.30–2.67), or high (>2.67) risk of fibrosis.

Statistical analysis

The statistical analysis was performed using SPSS version 23.0 software (IBM Corp., Armonk, NY, USA). In descriptive statistics, normal distribution is determined using the One-Sample Kolmogorov–Smirnov test. The continuous

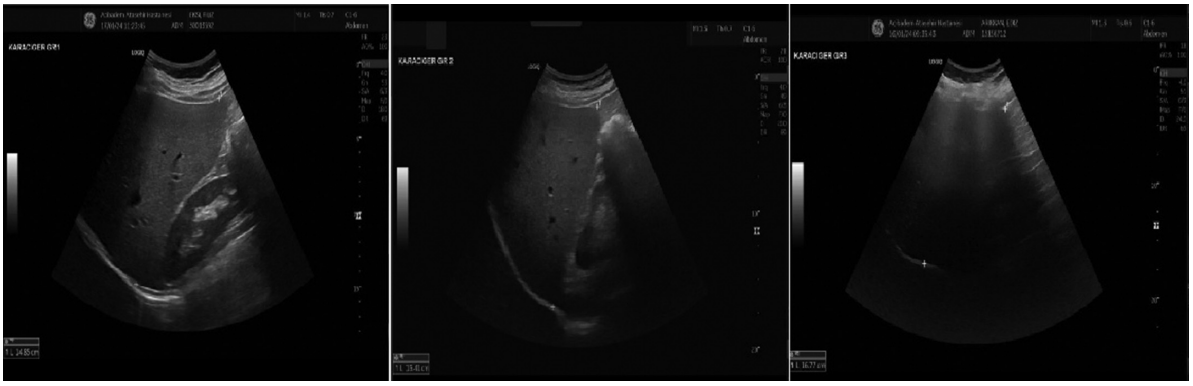

Image 1. Grade I (diffusely increased hepatic echogenicity but periportal and diaphragmatic echogenicity is still appreciable); grade II (diffusely increased hepatic echogenicity obscuring periportal echogenicity but diaphragmatic echogenicity is still appreciable); grade III (diffusely increased hepatic echogenicity obscuring periportal as well as diaphragmatic echogenicity).

Table 1. Demographic and clinical properties of the groups.

	Hepatosteatosis			Comparisons of groups (*P* values)		
	Stage 1	Stage 2	Stage 3	Stages 1 & 2	Stages 1 & 3	Stages 2 & 3
Age (years)	48.9 ± 9.2	48 ± 8.6	48.1 ± 9.2	0.129	0.284	0.921
Sex				0.005	0.042	0.637
Female	63 (23.6)	41 (14.3)	31 (15.9)			
Male	204 (76.4)	245 (85.7)	164 (84.1)			
Co-morbidities						
Hyperlipidemia	172 (64.4)	183 (64)	142 (72.8)	0.915	0.056	0.042
Dyslipidemia	258 (96.6)	277 (96.9)	190 (97.4)	0.882	0.617	0.709
Hyperglycemia	74 (27.7)	111 (38.9)	115 (59)	0.005	<0.001	<0.001
Diabetes mellitus	10 (3.7)	9 (3.1)	28 (14.4)	0.699	<0.001	<0.001
Hypertension	25 (9.4)	43 (15)	40 (20.6)	0.042	0.001	0.112
Dyspnea with effort	6 (2.3)	10 (3.5)	20 (10.4)	0.389	<0.001	0.002
Cerebrovascular disease	4 (1.5)	8 (2.8)	9 (4.6)	0.298	0.046	0.287
Physical condition						
Weight (kg)	83.3 ± 12.7	87.7 ± 12	97.3 ± 16.2	<0.001	<0.001	<0.001
Height (m)	1.71 ± 0.2	1.72 ± 0.22	1.73 ± 0.2	0.102	0.008	0.183
BMI (kg/m^2)	48.8 ± 2412	28.8 ± 3.4	31.9 ± 6.2	<0.001	<0.001	<0.001

Values are given as n (%) or mean ± SD. Statistical analyses were conducted using the Mann–Whitney U-test for continuous variables and chi-square test for categorical variables.
BMI, body mass index.

variables are expressed as mean ± standard deviation, while categorical variables are expressed in numbers and percentages. The Mann–Whitney U-test was used to compare the continuous variables of the two groups. Differences between categorical variables were calculated using the chi-square test. $P = 0.05$ was considered statistically significant.

Results

The demographic and clinical properties are given in Table 1. The participants were divided into three groups according to the ultrasound classification for hepatosteatosis: stage 1, 2, and 3. The median ages of the three hepatosteatosis groups were similar. Male ratio is greatest in the stage 2 hepatosteatosis group followed by the stage 3 hepatosteatosis group and finally the stage 1 hepatosteatosis group.

Hyperglycemia and diabetes mellitus were statistically associated with stage of hepatosteatosis ($P < 0001$). In addition, weight and body mass index (BMI) were also statistically associated with stage of hepatosteatosis ($P < 0.001$).

PLR values were highest in patients with stage 1 hepatosteatosis and progressively decreased in stages 2 and 3, with the lowest values observed in stage 3. The reduction in PLR was statistically significant when comparing stage 1 to stage

Table 2. Laboratory properties of the groups.

	Hepatosteatosis			Comparisons of groups (*P* values)		
	Stage 1	Stage 2	Stage 3	Stages 1 & 2	Stages 1 & 3	Stages 2 & 3
Laboratory results						
Platelets	2505 ± 55.8	251 ± 54.8	2596 ± 65	0.600	0.104	0.184
Lymphocytes	3.37 ± 14	3.9 ± 29.61	2.39 ± 0.68	0.164	<0.001	<0.001
PLR	1254 ± 42.69	1225 ± 38.48	115,26 ± 37.37	0.232	0.003	0.037
AST	21.2 ± 11.4	21.6 ± 8.2	27.7 ± 16.1	0.031	<0.001	<0.001
ALT	34.6 ± 17.4	41.7 ± 19.2	55.8 ± 31.9	<0.001	<0.001	<0.001
*FIB-4 index**	0.77 ± 0.43	0.69 ± 0.3	0.75 ± 0.38	0.163	0.783	0.313
<1.3 (low risk)	205 (91.1)	240 (95.2)	155 (90.6)	0.109	0.894	0.062
1.3–2.67 (moderate risk)	18 (8)	12 (4.8)	16 (9.4)			
>2.67 (high risk)	2 (0.9)	–	–			

Values are given as n (%) or mean ± SD. Statistical analyses were conducted using the Mann–Whitney U-test for continuous variables and chi-square test for categorical variables.
*FIB-4 index = (age*AST)/(platelet count*$\sqrt{}$ALT).
PLR, platelet to lymphocyte ratio.

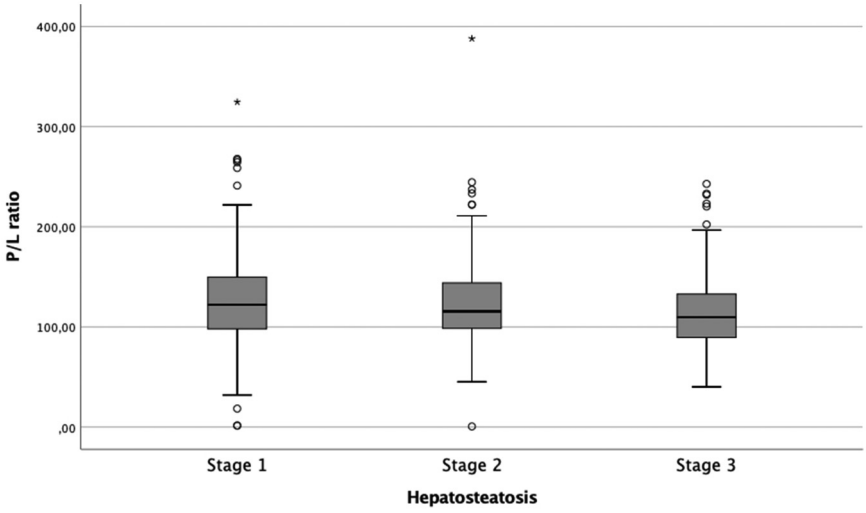

Fig. 1. The box-plot graphic of platelet to lymphocyte ratio in different stages of hepatosteatosis.

3 ($P = 0.003$) and stage 2 to stage 3 ($P = 0.037$). However, no significant difference was observed between stages 1 and 2, with the gap widening as steatosis severity increased. In addition, the FIB-4 index, an indicator of liver fibrosis, was calculated and found to be similar across all three groups (Table 2).

The box-plot graphic of PLR in the three hepatosteatosis groups is shown in Fig. 1.

Discussion

The aim of the present study was to investigate how PLR impacts the stages of hepatosteatosis on ultrasound. According to our analyses, platelet values increased slightly from stage 1 to 3 in hepatosteatosis, but this was not found to be statistically significant. The decrease in lymphocyte count varied between the first two stages and the third stage, with the difference being statistically significant ($P < 0001$). A low lymphocyte count indicates increased inflammation. The PLR value can be considered a sensitive indicator that is independent of sex and other laboratory markers. This focus on hepatosteatosis staging sets our study apart from others, offering a fresh perspective on how systemic inflammation and immune response, as reflected by PLR, are involved in the progression of fatty liver disease.

The FIB-4 index, a non-invasive scoring system, incorporating routine blood test results, patient age, and platelet count, was calculated to assess liver fibrosis. Notably, FIB-4 values did not show significant differences across the stages of hepatosteatosis. This suggests that

hepatosteatosis may be associated a reversible condition rather than one necessarily progressing to fibrosis.

Hepatosteatosis typically progresses through various stages, ranging from simple non-alcoholic fatty liver (NAFL) to non-alcoholic steatohepatitis (NASH) and, in some cases, ultimately to cirrhosis. Inflammation plays a crucial role in the progression of hepatosteatosis (12).

NAFLD is classified into two stages: NAFL, representing the early stage, and NASH, the more advanced stage. Although liver biopsy remains the gold standard for distinguishing between NAFL and NASH, its invasive nature, associated risks, and high cost (13) have driven the demand for non-invasive alternatives. Blood tests offer a practical solution due to their ease of use, safety, convenience, and affordability.

The role of platelets in inflammation extends beyond their traditional function in coagulation. Studies have shown that platelets express surface receptors capable of recognizing pathogens and immune complexes, underscoring their vital role in immune and inflammatory responses. The interaction between platelet P-selectin and leukocyte P-selectin ligand-1 facilitates platelet–leukocyte aggregation, forming a crucial link between leukocytes and the endothelium (14–16). An increased platelet count together with a decreased lymphocyte count is indicative of inflammation. The observed decrease in PLR makes this parameter a valuable tool in this context (17–19).

A cross-sectional study conducted in China examined the relationship between the PLR and hepatosteatosis. The study included two groups: 3413 non-NAFLD participants (median age = 39 years) and 1085 NAFLD participants (median age = 44 years). They identified an inflection point for PLR at 42.29. The study found that BMI, ALT, glucose, and hypercholesterolemia were positively associated with NAFLD, while PLR was negatively associated with NAFLD (9). These findings are similar to ours; however, our study provides more detailed insights into the variations in PLR across the different stages of hepatosteatosis.

Another study from Mexico was conducted with 278 participants and divided into four groups: group 1 = non-obese without NAFLD; group 2 = non-obese with NAFLD; group 3 = obese without NAFLD; and group 4 = obese with NAFLD. The study also showed that PLR is a novel parameter inversely correlated with NAFLD in non-obese patients (20).

A retrospective cohort study of untreated chronic HBV infection, HBV-related cirrhosis (HBV-CC), and HBV-related HCC patients found that PLR was significantly lower in HBV-CC patients than in other patients. The relationship between the PLR and both serum HBV-DNA and serum HBeAg across the different phases of chronic HBV infection was revealed. These preliminary findings suggest that the PLR may be used for the prediction of chronic HBV infection outcomes (6).

The relationship between PLR and HCC was analyzed in a meta-analysis of 21 studies, which showed that a higher PLR was associated with worse overall survival (OS), relapse-free/disease-free survival, and progression-free survival in these patients. PLR had poor prognostic value for survival in HCC patients when the cutoff value was >150. Chronic HBV infection is responsible for 50%–80% of HCC cases, and in this subgroup, a higher PLR was confirmed to be associated with poorer OS compared to other HBV subgroups. This may be due to chronic HBV being the major pathogen for HCC in China (7).

The FIB-4 index as a predictor of fibrosis has been examined in many studies. In our study, the FIB-4 index was similar across hepatosteatosis stages, which can be explained by the absence of fibrosis in these patients. This may serve as evidence that they were still in the reversible stage (9).

The present study has some limitations. The main limitation is that it is a retrospective cross-sectional analysis that does not allow for tracking changes over time. In addition, since the participants were volunteers, we were unable to obtain a liver biopsy or fibro scan data to assess the exact liver status of these patients. Furthermore, data on epidemiological patterns such as lifestyle, environment, and economy as predictors of NAFLD progression are not available for the participants.

An unexpected result of our study is was the decrease in lymphyocyte count as steatosis progressed. This suggests that not only immune system cells but also platelets play a role as indicators of inflammation.

In conclusion, we found that PLRs vary across the stages of hepatosteatosis.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

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