MedCrave
Step into the World of Research
Journal of Bacteriology & Mycology: Open access

Research Article
Open Access
CrossMark

Correlation between ASAT, ALAT, and gamma-GT levels and hepatic steatosis assessed by ultrasound at Les Promoteurs de la Bonne Santé medical center, Yaoundé, Cameroon

Abstract

Background: Fatty liver disease represents a growing public health problem in sub-Saharan Africa. Investigating its association with common liver biochemical markers is a fundamental diagnostic challenge for ultrasound, as it provides a linear correlation with disease severity.

Objective: To evaluate the correlation between serum levels of AST, ALT, and gamma-GT and the grade of hepatic steatosis determined by abdominal ultrasound.

Materials and methods: A cross-sectional analytical study involving 47 patients seen at the Les Promoteurs de la Bonne Santé Medical Center in Yaoundé between January and August 2024. Enzyme assays (AST, ALT, GGT) were performed using a kinetic spectrophotometric method. The grade of steatosis was assessed by abdominal ultrasound according to the international classification (grades 1, 2, and 3).

Results: Women made up the majority of the study population (55.3%), and the mean age was 51.8 ± 14.2 years. Grade 1 steatosis was the most common (63.8%), followed by grade 2 (27.7%) and grade 3 (8.5%). No statistically significant correlation was found between AST levels ($r = -0.082$; $p = 0.583$), ALT levels ($r = -0.128$; $p = 0.390$), or GGT levels ($r = -0.002$; $p = 0.990$) and the ultrasound grade of steatosis. The Kruskal-Wallis test confirmed the absence of significant differences between grades.

Conclusion: In our cohort, transaminases and GGT are not reliable markers for predicting the grade of hepatic steatosis. Ultrasound remains the first-line diagnostic test of choice.

Keywords: hepatic steatosis, ultrasound

Volume 14 Issue 2 - 2026

Kuiate David,[1,2,3,6] Kougoum Takam Natacha,[2] Nanfack Fanny Nadege,[2] Fomekong Dieuseul Lothner,[2,4] Foundikou Vessah Dayib,[2] Apouakone Mefire Aicha,[2] Pountougnogni Marie Laure,[7] Nzaneliagnigni Georgette,[7] Nana Yasmine Laurie[8]

[1]International School of Health and Medical Sciences, Kesmonds International University, Cameroon
[2]Department of Radiology and Medical Imaging, Les Promoteurs de la Bonne Santé, Cameroon
[3]Faculty of Medicine and Biomedical Sciences, University of Yaoundé, Cameroon
[4]Health Without Borders" School in Sanaga Maritime-Edéa, Cameroon
[5]Cardiology Department, "Les Promoteurs de la Bonne Santé" Medical Center, Cameroon
[6]Department of Biomedical Sciences, University of Ngaoundéré, Cameroon
[7]Laboratory and Medical Analysis Department, "Les Promoteurs de la Bonne Santé" Medical Center, Cameroon
[8]Le Samaritain Plus" Medical and Surgical Center, Cameroon

Correspondence: Kuiate David, International School of Health and Medical Sciences, Kesmonds International University, Cameroon, Tel 674 876 897

Received: May 27, 2026 | **Published:** June 05, 2026

Abbreviations: AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase

Introduction

Elevated liver function tests are a common finding in outpatient settings, estimated to occur in 1–4% of an asymptomatic population.[1] Liver test values fluctuate physiologically depending on age, sex, pregnancy, blood type, postprandial status, and physical exercise (EASL). These variations require clinical interpretation contextualized by local epidemiology and the profile of the liver test abnormalities.

Hepatic steatosis, characterized by excessive lipid accumulation in hepatocytes, is a growing health problem in Cameroon. Often asymptomatic, this condition can progress to steatohepatitis, fibrosis, and cirrhosis.[2] Despite its clinical significance, there is no indication for routine screening of liver parameters in the absence of risk factors or suggestive symptoms.

Liver enzymes, particularly AST (Aspartate Aminotransferase), ALT (Alanine Aminotransferase), and Gamma-GT (Gamma-glutamyltransferase), are standard biological markers used to assess liver damage. However, their exact correlation with hepatic steatosis detected by ultrasound remains variable across studies and populations.[1] Hepatic steatosis is frequently associated with a moderate and chronic elevation of transaminases, primarily ALAT, reflecting inflammation (steatohepatitis) or liver cell damage caused by fat accumulation.

This study aims to determine whether AST, ALT, and gamma-GT levels are significantly correlated with the presence and degree of hepatic steatosis in patients seen at the Les Promoteurs de la Bonne Santé Medical Center in Yaoundé.

Materials and methods

This is a cross-sectional study with an analytical focus, conducted at the "Les Promoteurs de la Bonne Santé" Medical Center, in the functional clinical laboratory and medical imaging departments equipped with a Mindray DC-80 Pro ultrasound machine, during the period from January to January 2024. We included all 47 adult and adolescent patients referred simultaneously for liver function tests and ultrasound evaluation, whose ultrasound diagnosis of hepatic steatosis was confirmed, and who signed an informed consent form. Excluded were patients with alcohol consumption exceeding 20 g/day (women) or 30 g/day (men), those undergoing hepatotoxic treatment or taking corticosteroids, and pregnant women. Serum levels of AST, ALT, and GGT were measured using the IFCC (International Federation

©2026 David et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

of Clinical Chemistry) kinetic spectrophotometric method on a semi-automated analyzer. The normal reference values used are: AST < 35 U/L (men) and < 31 U/L (women); ALT < 45 U/L (men) and < 34 U/L (women); GGT < 55 U/L (men) and < 38 U/L (women).

The abdominal ultrasound was performed after the patient had fasted for at least 6 hours, by a physician trained in gastrointestinal ultrasound. Steatosis was graded according to the commonly used international classification: **Grade 1 (mild)**: mild hepatic hyperechogenicity with preserved portal vessels; **Grade 2 (moderate)**: marked hyperechogenicity with moderate attenuation and partial visualization of vascular structures; **Grade 3 (severe)**: intense hyperechogenicity with marked posterior attenuation and obliteration of vessels.

The data were analyzed using SPSS software version 25.0, and the threshold for statistical significance was set at $p < 0.05$.

Results

A total of 47 patients meeting the inclusion criteria were included. Table 1 presents the general characteristics of the sample.

Table 1 General characteristics of the study population

Variable	Number / Value	Percentage / SD	Min – Max
Total number of patients	47	—	—
Female	26	55.3%	—
Male	21	44.7%	—
Average age (years)	51.8	± 14.2	16–90
ASAT — mean (U/L)	20.3	± 7.7	4.4 – 37.9
ALAT — mean (U/L)	23.6	± 19.1	2.7 – 101.9
GGT — mean (U/L)	47.4	± 86.3	9.6 – 571.2
Grade 1 steatosis	30	63.8%	—
Grade 2 steatosis	13	27.7%	—
Grade 3 steatosis	4	8.5%	—

General characteristics of the study population

The population was predominantly female (55.3%) with a mean age of 51.8 years, reflecting the typical profile of patients seeking liver function testing in Central Africa. Mild steatosis (grade 1) accounted for nearly two-thirds of cases (63.8%), moderate steatosis affected more than a quarter of patients (27.7%), and severe steatosis was present in a minority of cases (8.5%). The median values for AST, ALT, and GGT were generally within normal limits, reflecting the often-asymptomatic nature of this condition from a biological standpoint.

Enzyme levels by grade of steatosis

Table 2 presents the mean values and standard deviations of liver enzymes for each grade of steatosis, as well as the results of the Kruskal-Wallis test.

Table 2 Enzyme levels by grade of hepatic steatosis

Parameter	Grade 1 (n=30)	Grade 2 (n=13)	Grade 3 (n=4)	p (Kruskal-Wallis)
Mean ASAT (U/L) ± SD	20.6 ± 8.0	19.5 ± 6.5	20.8 ± 11.2	0.846
ALAT (U/L) mean ± SD	26.1 ± 23.0	20.0 ± 8.1	16.6 ± 5.3	0.571
GGT (U/L) mean ± SD	55.3 ± 106.8	35.2 ± 23.0	27.2 ± 7.6	0.970
Mean age (years)	50.0 ± 13.5	50.4 ± 11.4	69.0 ± 19.2	0.230

The mean levels of the three enzymes do not increase in a linear fashion as the grade of steatosis worsens. For ALT and GGT, an inverse trend is observed (higher values in grade 1 than in grade 3), although this is not statistically significant. This absence of a dose-response gradient is an important finding. The Kruskal-Wallis test reveals no statistically significant differences between the groups for any of the three enzymes ($p > 0.05$ in all cases).

Correlations between liver enzymes and steatosis grade

Table 3 presents the Spearman correlation coefficients between each liver enzyme and the ultrasound grade of steatosis.

Table 3 Spearman's correlations between liver enzymes and the grade of steatosis

Biological parameter	Spearman's r	p-value	95% CI	Significance
ASAT vs. Steatosis Grade	-0.082	0.583	[-0.36; 0.21]	NS
ALAT vs. Steatosis Grade	-0.128	0.390	[-0.40; 0.17]	NS
GGT vs. Steatosis Grade	-0.002	0.990	[-0.29; 0.29]	NS

Citation: David K, Natacha KT, Nadege NF, et al. Correlation between ASAT, ALAT, and gamma-GT levels and hepatic steatosis assessed by ultrasound at Les Promoteurs de la Bonne Santé medical center, Yaoundé, Cameroon. *J Bacteriol Mycol Open Access.* 2026;14(2):52–55. DOI: 10.15406/jbmoa.2026.14.00430

None of the three enzymatic biomarkers showed a statistically significant correlation with the grade of hepatic steatosis assessed by ultrasound. The Spearman coefficients are all close to zero or even slightly negative, indicating the absence of any linear or monotonic relationship between these variables. These results suggest that biological hepatic cytolysis does not correlate with the ultrasound-assessed severity of steatosis in our population.

Inter-enzyme correlations

Table 4 presents the Pearson correlations between the three enzymes studied.

Table 4 Pearson correlations between liver enzymes

Enzyme pair	Pearson's r	p-value	R^2	Significance
ASAT vs. ALAT	0.066	0.660	0.004	NS
ASAT vs GGT	-0.271	0.065	0.073	NS (trend)
ALAT vs GGT	0.245	0.097	0.060	NS (trend)

The inter-enzyme correlations are all weak and not significant at the 5% level. Marginal trends are observed between ALAT and GGT ($r = 0.245$; $p = 0.097$), and between AST and GGT ($r = -0.271$; $p = 0.065$), which could reach statistical significance with a larger sample size. The absence of a strong correlation between AST and ALT suggests partially independent hepatocellular injury mechanisms in this context.

Prevalence of enzyme abnormalities

Table 5 presents the proportion of patients with enzyme levels above normal reference values.

Table 5 Prevalence of enzyme abnormalities

Enzyme	Normal values (U/L)	High n	Total %	Median value (U/L)
AST	M < 35; F < 31	3 / 47	6.4%	18.0
ALAT	M < 45; F < 34	4 / 47	8.5%	19.4
GGT	M < 55; F < 38	8 / 47	17.0%	26.0

The vast majority of patients have normal enzyme levels: only 6.4% have elevated AST, 8.5% have elevated ALT, and 17.0% have elevated GGT. This finding strikingly illustrates the phenomenon of "fatty liver with normal laboratory findings," frequently reported in the international literature and confirmed here in an African context.

Discussion

The results of our study reveal a lack of statistically significant correlation between serum levels of AST, ALT, and GGT and the ultrasound grade of hepatic steatosis within our population in Yaoundé. This result, although it may seem surprising at first glance, is actually consistent with numerous international studies that highlight the same diagnostic paradox.

It is essential to understand why this finding is biologically consistent. The transaminases AST and ALT are markers of hepatocyte lysis, that is, the destruction of liver cells. However, simple hepatic steatosis, without inflammation or fibrosis, can exist without causing significant lysis. Hepatic lipogenesis and the accumulation of triglycerides within hepatocytes are not, in themselves, cytolytic processes. It is only when steatosis progresses to non-alcoholic steatohepatitis (NASH), with activation of inflammatory and oxidative pathways, that cytolysis sets in and transaminases rise significantly.

These observations are echoed in the work of Mofrad et al.,[3] who demonstrated that in patients with histologically confirmed steatosis, transaminases remained within normal limits in more than 59% of cases of mild NAFLD. Similarly, the study by Siddiqui et al.[4] showed that among patients with proven steatohepatitis, a majority had normal or only very slightly elevated transaminases, thereby calling into question the absolute diagnostic value of these enzymes in this context.

In sub-Saharan Africa, the few available studies corroborate this trend. Ndong et al.,[5] in a study conducted in Senegal, found no significant correlation between ALT and the ultrasound grade of steatosis in a series of 89 patients. Similarly, Tiomo et al.[6] in Cameroon observed that more than 70% of patients with ultrasound-detected steatosis had strictly normal liver enzymes, highlighting the need for multimodal diagnostic approaches in our settings.

GGT warrants special attention. This enzyme is often considered a more sensitive marker of NAFLD than transaminases, particularly because of its role in glutathione metabolism and hepatic oxidative stress. Recent meta-analyses, including that by Karajamaki et al.,[7] have shown that elevated GGT levels constitute an independent risk factor for NAFLD and progression to fibrosis. In our study, although GGT was most frequently above normal (17% of patients), its correlation with ultrasound grade remained null ($r = -0.002$; $p = 0.990$). This discrepancy may be explained by the fact that elevated GGT levels may reflect other metabolic processes (metabolic syndrome, insulin resistance) independent of hepatic fat content.[8,9]

The trend toward decreasing enzyme levels as the grade of steatosis worsens, although not statistically significant,[10] also warrants consideration. This paradoxical phenomenon could be explained by a lower representation of severe forms in our sample ($n = 4$ for grade 3), which considerably limits the statistical power of intergroup comparisons for this subgroup.[11]

From an epidemiological perspective, our population exhibits characteristics consistent with African data: a predominance of women, middle-aged adults, and a majority with mild steatosis. The slight over representation of women is consistent with the known role of sex hormones in hepatic lipid metabolism, particularly in postmenopausal women.[12-15]

Conclusion

This analytical cross-sectional study conducted at the Les Promoteurs de la Bonne Santé Medical Center in Yaoundé showed no statistically significant correlation between serum levels of AST, ALT, and gamma-GT and the ultrasound grade of hepatic steatosis. The vast majority of patients (more than 83%) had strictly normal enzyme levels despite the presence of steatosis confirmed by imaging. This result reinforces a fundamental clinical conviction: normal liver function test results do not rule out significant hepatic steatosis. Abdominal ultrasound therefore remains indispensable and irreplaceable for the screening, diagnosis, and monitoring of this condition in our setting. It should not be contingent upon prior elevation of liver enzymes. Large-scale, multicenter studies incorporating innovative non-invasive biomarkers and comprehensive clinical and metabolic data are strongly desired to help develop diagnostic recommendations tailored to our epidemiological context.

Funding

This study received no external funding.

Acknowledgments

None

Copyright: ©2026 David et al.

Conflicts of interest

The authors declare that they have no conflicts of interest related to this article.

References

1. Chalasani N, Younossi Z, Lavine JE, et al. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the AASLD. *Hepatology*. 2018;67(1):328–357.
2. Bedossa P, FLIP pathology consortium. utility and appropriateness of the fatty liver inhibition of progression (FLIP) algorithm and steatosis, activity, and fibrosis (SAF) score. *Hepatology*. 2014;60(2):565–575.
3. Mofrad P, Contos MJ, Haque M, et al. Clinical and histologic spectrum of nonalcoholic fatty liver disease associated with normal ALT values. *Hepatology*. 2003;37(6):1286–92.
4. Siddiqui MS, Sterling RK, Luketic VA, et al. Association between high–normal alanine aminotransferase levels and risk factors for metabolic syndrome in patients with NAFLD. *Gastroenterology*. 2013;144(7):1427–1435.
5. Ndong M, Diallo A, Bassene ML, et al. Biological profile of patients with hepatic steatosis in Dakar. *Rev Afr Med Interne*. 2019;6(2):12–18.
6. Tiomo R, Mbeh G, Moby C, et al. Non–alcoholic fatty liver disease in Cameroon: epidemiological, clinical, and paraclinical aspects. *Health Sci Dis*. 2021;22(4):45–52.
7. Käräjämäki AJ, Pätsi OP, Savolainen M, et al. Non–alcoholic fatty liver disease as a predictor of atrial fibrillation in a middle–aged population (OPERA Study). *PLoS One*. 2016;11(10):e0165855.
8. Younossi ZM, Koenig AB, Abdelatif D, et al. Global epidemiology of nonalcoholic fatty liver disease- a meta–analytic assessment of prevalence, incidence, and outcomes. *Hepatology*. 2016;64(1):73–84.
9. Ekstedt M, Hagström H, Nasr P, et al. Fibrosis stage is the strongest predictor for disease–specific mortality in NAFLD after up to 33 years of follow–up. *Hepatology*. 2015;61(5):1547–1554.
10. Castera L, Friedrich–Rust M, Loomba R. Noninvasive assessment of liver disease in patients with nonalcoholic fatty liver disease. *Gastroenterology*. 2019;156(5):1264–81.
11. Pekala A, Gwizdek K, Chrobak–Bień J, et al. Comparison of results of laboratory tests and ultrasound examination in the diagnosis of fatty liver disease. *Przegl Gastroenterol*. 2015;10(3):159–164.
12. Angulo P, Hui JM, Marchesini G, et al. The NAFLD fibrosis score: a noninvasive system that identifies liver fibrosis in patients with NAFLD. *Hepatology*. 2007;45(4):846–854.
13. European association for the study of the liver, EASD, EASO. EASL–EASD–EASO clinical practice guidelines for the management of non–alcoholic fatty liver disease. *J Hepatol*. 2016;64(6):1388–402.
14. Browning JD, Szczepaniak LS, Dobbins R, et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. *Hepatology*. 2004;40(6):1387–1395.
15. Szczepaniak LS, Nurenberg P, Leonard D, et al. Magnetic resonance spectroscopy to measure hepatic triglyceride content: prevalence of hepatic steatosis in the general population. *Am J Physiol Endocrinol Metab*. 2005;288(2):E462–E468.

Citation: David K, Natacha KT, Nadege NF, et al. Correlation between ASAT, ALAT, and gamma-GT levels and hepatic steatosis assessed by ultrasound at Les Promoteurs de la Bonne Santé medical center, Yaoundé, Cameroon. *J Bacteriol Mycol Open Access*. 2026;14(2):52–55. DOI: 10.15406/jbmoa.2026.14.00430