Research Article
Open Access
CrossMark

Assessing the predictive accuracy of the aMAP risk score for Hepatocellular Carcinoma

Abstract

Introduction: Hepatocellular carcinoma (HCC) is a significant complication of cirrhosis, associated with high morbidity and mortality rates. Various scoring systems have been developed to predict the risk of HCC development, including the aMAP score, which was developed and validated in 2020. This study aims to evaluate the efficacy of the aMAP score in predicting the onset of hepatocellular carcinoma.

Patients and methods: We conducted a retrospective analysis of 62 cirrhotic patients monitored in our department between 2006 and 2022. We collected data on demographics, clinical parameters, cirrhosis status, HCC imaging results, and alpha-fetoprotein levels. The aMAP score (ranging from 0 to 100) was calculated based on age, sex, albumin-bilirubin levels, and platelet count. Data were analyzed using SPSS software version 26.

Results: A total of 62 patients were analyzed, with a male-to-female ratio of 1.36. The mean age was 64 ± 12.7 years. The primary causes of cirrhosis included hepatitis C virus (35.5%), hepatitis B virus (30.6%), metabolic dysfunction-associated steatotic liver disease (6.5%), and indeterminate origins (27.4%). During follow-up, 45.2% (n=28) of patients developed HCC, with a mean time to onset of 12 months. Imaging revealed a single nodule in 64.7% (n=11) of cases. The mean aMAP score was 64.54 ± 9.08. The aMAP score was significantly higher in the HCC group compared to the non-HCC group. The predictive performance of the aMAP score for HCC development was significant, with an area under the receiver operating characteristic curve (AUROC) of 0.754 ($p < 0.001$). The optimal cut-off score was determined to be 57.5, yielding a sensitivity of 89% and a specificity of 56%.

Conclusion: The aMAP score is an accurate and user-friendly tool for predicting hepatocellular carcinoma in patients with cirrhosis. Its application in clinical practice may enhance surveillance, improve early detection of HCC, and potentially reduce mortality rates associated with this malignancy.

Keywords: aMAP score, hepatocellular carcinoma, cirrhosis, risk prediction, surveillance, diagnostic accuracy

Volume 16 Issue 4 - 2025

E Slama,[1] S Ben Hamida,[2] H Elloumi,[2] I.Cheikh[2]
[1]University of medicine of Tunis El MANAR, Tunisia
[2]Department of gastroenterology, Universitary hospital of Bizerte, Tunisia

Correspondence: E Slama, University of medicine of Tunis El MANAR, Tunisia

Received: July 4, 2025 | **Published:** August 23, 2025

Introduction

Hepatocellular carcinoma (HCC) represents a major global health burden, being the most common primary malignancy of the liver and a leading cause of cancer-related mortality worldwide. It typically develops in the setting of chronic liver disease, particularly cirrhosis, regardless of etiology. The high morbidity and mortality rates associated with HCC underscore the critical need for timely detection and effective risk stratification strategies.

Over the years, several risk prediction models have been proposed to identify individuals at increased risk of HCC development. Earlier tools such as the GAG-HCC and CU-HCC scores were valuable in their time but were often limited by complexity and a lack of external validation across varied populations, reducing their clinical utility in diverse real-world settings.

In response to the need for a more accessible and broadly applicable tool, Fan et al.[1] introduced the aMAP score: a simple, non-invasive model designed to predict HCC risk in patients with chronic liver disease. The aMAP score incorporates four clinical parameters: age, sex, albumin-bilirubin (ALBI) grade, and platelet count. Its primary objective is to facilitate stratification of HCC risk, particularly among patients with cirrhosis, in order to guide surveillance strategies and optimize resource allocation.

Aims: Despite its promising initial validation, the aMAP score has undergone limited evaluation in real-world clinical settings, especially in ethnically and geographically diverse populations. Therefore, the present study aims to assess the predictive accuracy of the aMAP score for HCC onset.

Patients and methods

Methods

Study design

This retrospective cohort study was conducted at the gastroenterology department of Bizerte's Universitary hospital over a 17-year period, from January 2006 to December 2022. The study was approved by the institutional review board (IRB), and all procedures conformed to ethical standards for human research.

Patient population

The study included patients with a confirmed diagnosis of cirrhosis based on imaging findings, histological analysis, or established clinical criteria. Inclusion criteria were: age ≥18 years, a confirmed diagnosis of cirrhosis, and a minimum follow-up duration of six months within the department. Patients were excluded if they had a prior diagnosis of hepatocellular carcinoma (HCC) at baseline, had incomplete clinical or laboratory data, or were lost to follow-up before six months. A total

of 62 patients met the inclusion criteria and were enrolled in the final analysis. Although the sample size is relatively limited, this study is intended as a pilot investigation, conducted under resource and data availability constraints.

Data collection

Clinical and demographic data were collected from patient records. The following parameters were recorded:

a) **Demographics:** Age and sex.

b) **Clinical characteristics:** Etiology of cirrhosis (classified as hepatitis C virus [HCV], hepatitis B virus [HBV], metabolic dysfunction-associated steatotic liver disease [MASLD], or indeterminate), liver function tests (including ALT, AST, bilirubin, albumin, INR), and alpha-fetoprotein (AFP) levels.

c) **Imaging findings:** Confirmation of HCC diagnosis was based on radiological criteria from contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI), and data on nodule size and characteristics were collected when available.

d) **aMAP score calculation:** The aMAP score was calculated for each patient using the original formula published by Fan et al., which incorporates age, sex, albumin-bilirubin (ALBI) grade, and platelet count. The score ranges from 0 to 100, with higher values indicating greater HCC risk.

Statistical analysis

Statistical analysis was performed using SPSS version 26. Descriptive statistics were used to summarize patient characteristics, with continuous variables expressed as mean ± standard deviation (SD) and categorical variables as percentages. Group comparisons between patients who developed HCC and those who did not were performed using the Student's t-test for continuous variables and the chi-square test for categorical variables. To assess the predictive performance of the aMAP score, receiver operating characteristic (ROC) curve analysis was conducted. The area under the ROC curve (AUROC) was calculated, along with sensitivity, specificity, and the optimal cut-off value determined using Youden's index. A p-value of less than 0.05 was considered statistically significant (Figure 1).

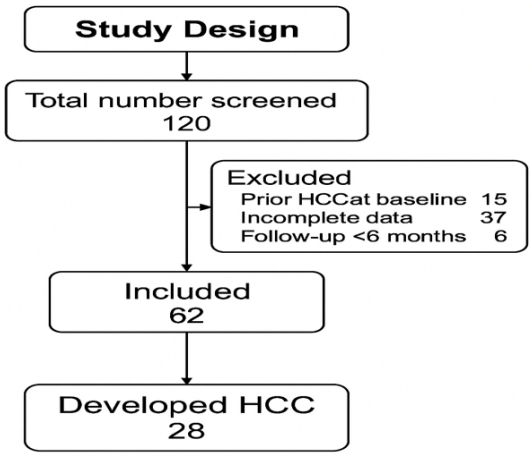

Figure 1 Study design.

Results

The study cohort included 62 patients with cirrhosis, comprising 36 men and 26 women (male-to-female ratio: 1.36), with a mean age of 64 ± 12.7 years. The etiologies of cirrhosis were hepatitis C virus (HCV) in 35.5% (n=22), hepatitis B virus (HBV) in 30.6% (n=19), metabolic-associated steatotic liver disease (MASLD) in 6.5% (n=4), and indeterminate in 27.4% (n=17). During the follow-up period, hepatocellular carcinoma (HCC) developed in 45.2% of patients (n=28), with a mean time to onset of 19 months [*16 months-33 months]*. Imaging data were available for all patients, among whom a single hepatic nodule was identified in 64.7% (n=11). The mean aMAP score for the overall cohort was 64.54 ± 9.08, and was significantly higher in patients who developed HCC *(68.85 ±7.67)* compared to those who did not *(61 ± 8.69*; $p < 0.001$) .The aMAP score demonstrated moderate predictive accuracy for HCC, with an area under the receiver operating characteristic curve (AUROC) of 0.754 (95% CI: [0.632-0.876]; $p < 0.001$). An optimal cut-off value of 57.5 was identified, yielding a sensitivity of 89% and a specificity of 56%.

A binary logistic regression analysis was conducted to identify factors associated with the development of hepatocellular carcinoma (HCC) in patients with cirrhosis. Variables included in the model were sex, age, aMAP score, alpha-fetoprotein (AFP), cirrhosis etiology, Model for End-Stage Liver Disease (MELD) score, and Child-Pugh score. Among these, the aMAP score demonstrated the strongest trend toward statistical significance (B = 0.289; p = 0.056), with an odds ratio (OR) of 1.335, suggesting a potential association between higher aMAP scores and increased risk of HCC. AFP levels also showed a non-significant trend toward association (B = 0.052; p = 0.097; OR = 1.053), indicating a modest increase in risk with higher AFP values. In contrast, no statistically significant associations were found for sex (p = 0.355), cirrhosis etiology (p = 0.604), MELD score (p = 0.484), Child-Pugh score (p = 0.573), or age (p = 0.163). These findings suggest that the aMAP score may represent a valuable tool for HCC risk stratification in cirrhotic patients, though additional studies are warranted to validate these results (Figure 2).

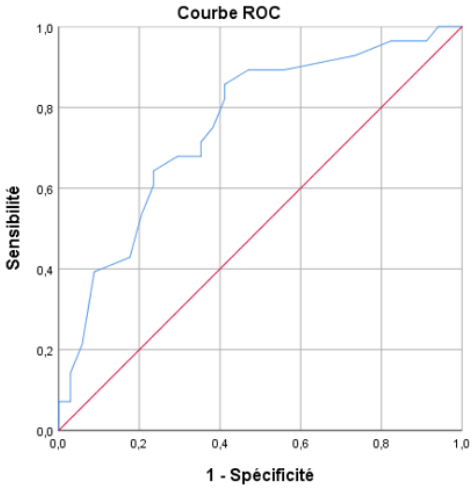

Figure 2 ROC curve showing AUROC of 0.754 with cut-off at 57.5.

Discussion

This study assessed the predictive value of the aMAP score for hepatocellular carcinoma (HCC) in a real-world cohort of patients with cirrhosis of diverse etiologies. Our findings demonstrate that the aMAP score is a potentially valuable tool for HCC risk stratification, showing moderate discriminatory performance (AUROC = 0.754; 95% CI: 0.632–0.876; $p < 0.001$), high sensitivity (89%), and a clinically relevant cut-off value of 57.5. Patients with

Citation: Slama E, Hamida SB, Elloumi H, et al. Assessing the predictive accuracy of the aMAP risk score for Hepatocellular Carcinoma. *Gastroenterol Hepatol Open Access*. 2025;16(4):106–109. DOI: 10.15406/ghoa.2025.16.00617

aMAP scores above this threshold were significantly more likely to develop HCC, reinforcing the potential utility of this tool in surveillance strategies for cirrhotic patients.

When compared with the original validation study by Fan et al.,[1] which re-ported an AUROC of approximately 0.82 in a large international cohort of over 17,000 patients with chronic hepatitis, the predictive performance in our study was somewhat lower. This discrepancy may reflect differences in study populations. Our cohort consisted exclusively of patients with cirrhosis and included a broader range of etiologies, with a relatively low prevalence of MASLD (6.5%). This contrasts with trends in Western countries, where MASLD is becoming the leading cause of chronic liver disease.[2,3] Other factors, such as differences in liver function reserve, fibrosis stage, or duration of follow-up, may also account for the variation in predictive accuracy. Nevertheless, the high sensitivity observed in our cohort supports the use of the aMAP score as a screening tool capable of identifying high-risk individuals, even if its specificity (56%) remains modest.

Our findings are consistent with several validation studies. Johnson et al.[4] validated the aMAP score in a large real-world cohort (n=3473), reporting a C-index of 0.81 and demonstrating its utility in identifying low-risk groups with HCC incidence <1 per 1000 person-years, aligning with our results for risk stratification. Similarly, Elgenidy et al.[5] conducted a meta-analysis showing a pooled sensitivity of 96.1% at a cut-off of 50, supporting the high sensitivity (89%) at our cut- off of 57.5 and reinforcing aMAP's role in surveillance. In post-resection HCC, Tsilimigras et al.[6] found that aMAP scores ≥64 predicted worse 5-year overall survival (54.3% vs. 66.5%), suggesting prognostic value beyond risk prediction, which complements our logistic regression findings (OR=1.335, p=0.056). Fan et al.[7] introduced enhanced models (aMAP-2/Plus) with AUROCs of 0.83–0.89, indicating potential improvements for cohorts like ours with high viral etiology prevalence. Chaiwiriyawong et al. [8] reported an AUROC of 0.777 at 5 years in chronic hepatitis B patients, similar to our 0.754, though CU-HCC outperformed aMAP in their study, a limitation we can explore. Gui et al.[9] validated aMAP in CHB-related cirrhosis on antiviral therapy (C-index 0.724), mirroring our results and supporting its applicability in treated viral cohorts. Lastly, Chen et al.[10] identified aMAP as a risk factor in intermediate-stage HCC, consistent with our finding of higher scores in the HCC group.

From a clinical standpoint, the aMAP score offers several key advantages. It is derived from routine demographic and biochemical parameters—age, sex, ALT, to bilirubin, and platelet count—making it a non-invasive, accessible, and cost- effective tool that can be easily implemented in daily practice. The identification of a practical cut-off (57.5) allows for stratified surveillance: patients with higher scores could benefit from closer follow-up using high-resolution imaging or adjunctive biomarker monitoring, such as alpha-fetoprotein (AFP), while those at lower risk may continue with standard biannual ultrasound surveillance. This risk-based approach may enhance early detection of HCC, reduce unnecessary procedures, and optimize resource allocation.

The role of Sirtuin 1 (SIRT1), a NAD+-dependent deacetylase, has been increasingly recognized in HCC and metabolic liver diseases. SIRT1 influences senescence, apoptosis, and HCC progression, with a dual role: protective in early stages by regulating cellular stress but pro-tumorigenic in advanced HCC via deacetylation of oncogenic factors like β-catenin.[11] Martins[12-14] high-lights SIRT1's relevance in appetite regulation, diabetes, and chronic disease, suggesting its potential as a diagnostic marker for HCC in metabolic contexts like MASLD. However, no studies directly compare plasma SIRT1 levels with aMAP for HCC risk stratification. While aMAP leverages simple clinical parameters for high sensitivity (89% in our cohort), SIRT1's diagnostic sensitivity/specificity re- mains less defined, likely serving better as a prognostic or treatment-response marker. Combining SIRT1 with aMAP could enhance specificity, a limitation in our study (56%), and warrants further investigation in larger cohorts.

One of the most compelling aspects of the aMAP score is its affordability and simplicity. It relies exclusively on inexpensive, commonly available laboratory data, making it particularly well suited for use in settings with limited access to advanced diagnostics or imaging. This broad applicability increases its potential for global adoption, especially in low- and middle-income countries where surveillance programs for HCC are often under-resourced. In addition, the algorithmic simplicity of the aMAP score facilitates its integration into electronic medical record (EMR) systems, where automated calculation and clinical alerts could standardize and streamline risk-based surveillance. Such integration may reduce variability in clinical practice, ensure timely follow-up, and minimize the burden on busy healthcare systems. As clinical decision-making becomes increasingly digitized, tools like aMAP can support more proactive and personalized care pathways.

Risk stratification using the aMAP score may influence clinical decision-making in meaningful ways. Patients with scores exceeding the threshold of 57.5 could be enrolled in intensified surveillance protocols, such as imaging every 3 to 4 months or the use of contrast-enhanced CT or MRI for better lesion detection. High-risk individuals might also warrant early referral to hepatology specialists or transplant centers for consideration of curative treatments, including resection, ablation, or transplantation. Conversely, patients with lower aMAP scores may remain on standard 6-monthly ultrasound surveillance, potentially reducing unnecessary investigations and associated healthcare costs. In this manner, the aMAP score enables a personalized, risk-adapted surveillance strategy that balances sensitivity with clinical and economic feasibility— ultimately aiming to improve outcomes by enabling earlier detection and intervention.

The strengths of this study include the use of real-world clinical data, a relatively long mean follow-up period of 19 months, and the inclusion of patients with varied etiologies of cirrhosis, reflecting the heterogeneity encountered in clinical practice. However, the study has several limitations. The small sample size limits statistical power and restricts generalizability. Its retrospective, single-center design may introduce selection and information bias. Additionally, the lack of external validation limits the extrapolation of our findings to broader populations. Future research should focus on validating these findings in larger, multicenter prospective studies.

Evaluating the performance of the aMAP score across different cirrhosis etiologies, fibrosis stages, and geographic populations will be essential. Furthermore, integrating the aMAP score with other biomarkers such as AFP or SIRT1, or with imaging findings, may enhance its specificity and refine risk prediction algorithms. Ultimately, a multiparametric approach may yield the most accurate and clinically actionable HCC risk stratification model (Figure 3).

Citation: Slama E, Hamida SB, Elloumi H, et al. Assessing the predictive accuracy of the aMAP risk score for Hepatocellular Carcinoma. *Gastroenterol Hepatol Open Access*. 2025;16(4):106–109. DOI: 10.15406/ghoa.2025.16.00617

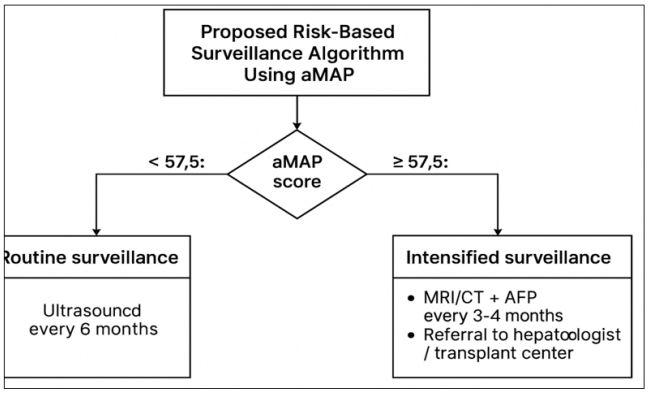

Figure 3 Proposed Risk-Based Surveillance Algorithm using aMAP.

References

1. Fan R, Papatheodoridis G, Sun J, et al. aMAP risk score predicts hepatocellular carcinoma development in patients with chronic hepatitis. *J Hepatol.* 2020;73(6):1368–1376.
2. Ekstedt M, Franzén LE, Mathiesen UL, et al. Long–term follow–up of patients with NAFLD and elevated liver enzymes. *Hepatology.* 2015;62(4):1265–1272.
3. Yki–Järvinen H. Non–alcoholic fatty liver disease as a cause and a consequence of metabolic syndrome. *Lancet Diabetes Endocrinol.* 2016;4(11):901–910.
4. Johnson PJ, Innes H, Hughes DM, et al. Evaluation of the aMAP score for hepatocellular carcinoma surveillance: a realistic opportunity to risk stratify. *Br J Cancer.* 2022;127:1263–1269.
5. Elgenidy A, Abubasheer TM, Odat RM, et al. Assessing the predictive accuracy of the aMAP risk score for hepatocellular carcinoma: diagnostic test accuracy and meta–analysis. *J Clin Exp Hepatol.* 2025;15(1):102381.
6. Tsilimigras DI, Endo Y, Ratti F, et al. The aMAP score predicts long–term outcomes after resection of hepatocellular carcinoma: a multi–institutional analysis. *HPB (Oxford).* 2024;26(4):541–547.
7. Fan R, Chen L, Zhao S, et al. Novel, high accuracy models for hepatocellular carcinoma prediction based on longitudinal data and cell–free DNA signatures. *J Hepatol.* 2023;79(4):933–944.
8. Chaiwiriyawong S, Assawasuwannakit S, Feuangwattana P, et al. Clinical utility of the aMAP score for predicting hepatocellular carcinoma development in patients with chronic hepatitis B. *Diagnostics (Basel).* 2024;14:1325.
9. Gui H, Huang Y, Zhao G, et al. External validation of aMAP hepatocellular carcinoma risk score in patients with chronic hepatitis B–related cirrhosis receiving ETV or TDF therapy. *Front Med (Lausanne).* 2021;8:677920.
10. Chen Y, Shi Y, Lu L, et al. The aMAP score is an independent risk factor for intermediate–stage hepatocellular carcinoma: a large retrospective cohort study. *J Cancer.* 2023;14(8):1272–1281.
11. Farcas M, Gavrea AA, Gulei D, et al. SIRT1 in the development and treatment of hepatocellular carcinoma. *Front Nutr.* 2019;6:148.
12. Martins IJ. Anti–aging genes improve appetite regulation and reverse cell senescence and apoptosis in global populations. *Adv Aging Res.* 2016;5:9–26.
13. Martins IJ. Single gene inactivation with implications to diabetes and multiple organ dysfunction syndrome. *J Clin Epigenet.* 2017;3(3):24.
14. Martins IJ. Sirtuin 1, a diagnostic protein marker and its relevance to chronic disease and therapeutic drug interventions. *EC Pharmacol Toxicol.* 2018;6(4):209–215.

Citation: Slama E, Hamida SB, Elloumi H, et al. Assessing the predictive accuracy of the aMAP risk score for Hepatocellular Carcinoma. *Gastroenterol Hepatol Open Access.* 2025;16(4):106–109. DOI: 10.15406/ghoa.2025.16.00617