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Original Research Article

Radiologic characteristics and prognostic outcomes in non-alcoholic fatty liver disease-related versus alcohol-related hepatocellular carcinoma: a comparative cohort study

Kuldeep Singh[1], Supriya Sundar Mishra[2]*, Anamika Shahi[2]

[1]Department of Gastroenterology, Apex Hospital, Jaipur, Rajasthan, India
[2]Department of Radiology, Institute of Medical Sciences and SUM Hospital, Bhubaneswar, Odisha, India

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***Correspondence:**
Dr. Supriya Sundar Mishra,
E-mail: radiodoc488@gmail.com

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) has emerged as one of the fastest-growing etiologies of hepatocellular carcinoma (HCC), while alcohol remains a dominant cause of cirrhosis and HCC in India. Differences in tumor morphology, biological behavior, and outcomes between these two etiologies remain inadequately defined in the Indian population. This study aims to compare radiologic features and prognostic outcomes in NAFLD-related and alcohol-related HCC patients.
Methods: This retrospective cohort study conducted at Apex Hospital included 50 consecutive HCC patients evaluated between September 2014 and October 2015. Etiology was classified as NAFLD (n=25) or alcohol-related liver disease (ALD) (n=25). All patients underwent contrast-enhanced computed tomography (CT) or MRI at presentation. Tumor morphology, liver imaging reporting and data system (LI-RADS) imaging features, vascular invasion, extrahepatic disease, and portal hypertension indicators were analyzed. Clinical parameters including serum alpha-fetoprotein (AFP), Child-Pugh and model for end-stage liver disease (MELD) scores, metabolic comorbidities, and treatment modalities were compared. Survival was assessed at 1 and 3 years.
Results: NAFLD-HCC patients were older (mean 62 vs 54 years), had higher rates of diabetes (68% vs 24%), and more often presented with solitary large lesions (mean size 6.1 cm vs 4.2 cm). Alcohol-HCC patients showed significantly higher rates of multifocal disease (56% vs 28%) and portal hypertension markers. Portal vein thrombosis was more common in alcohol-HCC (36% vs 16%). One-year survival was higher in the NAFLD group (68% vs 40%; $p<0.05$). Three-year survival remained better in NAFLD-HCC (24% vs 8%).
Conclusions: NAFLD-and alcohol-related HCC represent distinct phenotypic and prognostic subtypes. NAFLD-HCC is characterized by large solitary tumors but better liver function and improved short-term survival, while alcohol-HCC exhibits more advanced cirrhosis, multifocal disease, and worse outcomes. Etiology-specific surveillance strategies are warranted.

Keywords: NAFLD, Hepatocellular carcinoma, Magnetic resonance imaging, Computed tomography, Treatment

INTRODUCTION

Hepatocellular carcinoma (HCC) ranks among the most lethal malignancies worldwide and is responsible for over 700,000 cancer-related deaths each year.[1] Historically, chronic infection with hepatitis B and C viruses has been the dominant etiological factor for HCC across most regions.[2] In recent years, however, a significant epidemiological transition has been observed, marked by a rapid increase in HCC arising from NAFLD. This shift is largely attributable to the global surge in obesity, type 2 diabetes mellitus, and metabolic syndrome.[3,4] India is

experiencing a parallel trend, with escalating rates of diabetes and central obesity contributing substantially to the growing burden of NAFLD and its hepatic complications.[5,6]

ALD remains highly prevalent in India due to increasing hazardous drinking patterns and early initiation of alcohol use.[7] ALD accounts for advanced cirrhosis, portal hypertension, and is an established risk factor for HCC.[8] Compared with NAFLD, alcohol-related HCC arises in younger individuals and is characterized by more advanced liver dysfunction at presentation.[9]

NAFLD-related HCC is unique because it can develop even in the absence of cirrhosis, with 20-30% of cases arising in non-cirrhotic livers.[10,11] Surveillance for NAFLD patients remains suboptimal given the challenges in identifying high-risk individuals.[12] In contrast, alcohol-related HCC almost always develops upon a cirrhotic background and is strongly associated with portal hypertension and hepatic decompensation.[13,14]

Radiologic imaging is fundamental to the diagnosis and staging of HCC. Multiphase contrast-enhanced CT and MRI have become the gold standard due to high sensitivity and specificity when interpreted using LI-RADS criteria.[15,16] Imaging features including arterial phase hyperenhancement, washout, capsule appearance, fat content, diffusion restriction, and macrovascular invasion are essential for diagnosis and prognostication.[17]

Emerging literature suggests that NAFLD-HCC and alcohol-HCC differ radiologically. NAFLD-HCC often presents as larger, solitary masses, frequently subcapsular, possibly due to delayed diagnosis in non-cirrhotic NAFLD.[18,19] Alcohol-HCC tends to present with multifocal lesions, reflecting advanced cirrhosis and widespread liver injury.[20] Portal hypertension indicators such as splenomegaly and portosystemic collaterals are more pronounced in alcoholic cirrhosis.[21]

Prognosis also differs by etiology. NAFLD-HCC patients typically have lower AFP levels, better-preserved liver function, and improved survival compared with patients with alcohol-related HCC.[22,23] Conversely, aggressive tumor biology, vascular invasion, and recurrent decompensation contribute to poorer outcomes in alcohol-related HCC.[24,25]

Despite these global observations, there is limited Indian data comparing radiologic and prognostic differences between NAFLD- and alcohol-related HCC. Regional differences in metabolic risk factors, alcohol consumption patterns, and surveillance practices warrant localized research.[26]

The present study aims to compare radiologic morphology, liver background features, vascular invasion, and short- and long-term survival outcomes between NAFLD-related and alcohol-related HCC in a tertiary care center in North India.

METHODS

Study design and setting

This retrospective observational cohort study was conducted at a tertiary care gastroenterology and hepatology center in Apex Hospital, Jaipur, India. Institutional ethical approval was obtained prior to data extraction.

Study population

A total of 50 consecutive patients diagnosed with HCC between September 2014 and October 2015 were included. Diagnosis was confirmed based on LI-RADS criteria using contrast-enhanced CT/MRI or histopathology when required.

Group classification

NAFLD-HCC (n=25)

Diagnosed based on imaging evidence of hepatic steatosis, metabolic risk factors (diabetes, obesity, dyslipidemia), and absence of significant alcohol intake.

Alcohol-HCC (n=25)

Based on history of harmful alcohol consumption (>40 g/day for men, >20 g/day for women) with clinical and radiologic evidence of alcoholic cirrhosis.

Exclusion criteria

Viral hepatitis (HBV/HCV), mixed etiologies, insufficient imaging data and prior locoregional therapy before baseline imaging were excluded from the study.

Radiologic assessment and clinical variables

All enrolled patients underwent comprehensive radiologic evaluation using either triphasic contrast-enhanced CT or MRI. Imaging findings were interpreted and categorized according to the LI-RADS criteria. Radiologic variables documented included the size of the tumor based on the largest lesion identified, the total number of hepatic lesions, and their anatomical location with respect to hepatic segments, lobar distribution, and subcapsular involvement. Key imaging characteristics such as arterial phase hyperenhancement, presence of washout, and capsule appearance were systematically assessed. Evidence of vascular invasion, particularly portal vein or other macrovascular involvement, was recorded, along with the presence of extrahepatic metastases. Background liver morphology was also evaluated, including features of cirrhosis such as

nodularity, hepatic steatosis, splenomegaly, and portosystemic collaterals.

Clinical variables collected included patient demographics such as age and sex, as well as relevant comorbidities including diabetes mellitus and obesity. Liver disease severity was assessed using the Child-Pugh classification and MELD scores.

Serum alpha-fetoprotein (AFP) levels were documented for all patients. Clinical evidence of hepatic decompensation, including the presence of ascites, hepatic encephalopathy, and episodes of variceal bleeding, was also recorded.

Outcome measures

The primary outcome measure of the study was the assessment of radiologic differences in tumor characteristics. Secondary outcome measures included overall survival at 1 year and 3 years following diagnosis.

Statistical analysis

Statistical analysis was performed using appropriate tests based on the distribution and nature of the data. Continuous variables were compared between groups using the independent *t*-test for normally distributed data, while the Mann-Whitney U test was applied for non-normally distributed variables. Categorical variables were analyzed using the Chi-square test or Fisher's exact test, as appropriate. Survival outcomes were evaluated using Kaplan-Meier survival curves, and differences between groups were assessed using the log-rank test.

RESULTS

Patients with NAFLD-related HCC were older (mean age ~62 vs ~54 years) and had a higher prevalence of metabolic risk factors, particularly diabetes (68% vs 24%). In contrast, alcohol-related HCC predominantly affected males and was associated with higher AFP levels and worse baseline liver function, with a greater proportion presenting with hepatic decompensation (Table 1).

Significant differences were noted in tumor morphology between groups. Mean tumor size was larger in NAFLD-HCC patients (6.1±2.4 cm) compared to alcohol-HCC patients (4.2±1.9 cm; $p<0.05$). Solitary tumors were more frequently seen in NAFLD-HCC (68% vs 36%; p=0.02), whereas multifocal tumors were significantly more common in alcohol-related HCC (56% vs 28%; p=0.03). Subcapsular tumor location was more frequently observed in NAFLD-related HCC. LI-RADS major imaging features were comparable between groups, with fat-containing lesions more common in NAFLD-HCC and diffusion restriction commonly seen in both etiologies (Table 2).

Alcohol-related HCC patients showed significantly more advanced cirrhosis on imaging, with nodular liver morphology present in 88% compared to 56% in the NAFLD group ($p<0.01$). Steatosis was significantly more common among NAFLD-HCC patients (72% vs 16%; $p<0.001$). Markers of portal hypertension-splenomegaly and portosystemic collaterals-were significantly more prevalent in alcohol-HCC patients (80% and 76%, respectively) than in the NAFLD-HCC group (44% and 40%; $p<0.01$ for both). Portal vein thrombosis (36% vs 16%) and macrovascular invasion (28% vs 12%) were more common in the alcohol-HCC group, though these differences did not reach statistical significance (Table 3).

TACE was the most frequently used initial treatment modality in both groups (60% NAFLD vs 72% alcohol; p=0.36). RFA was performed more often in NAFLD-HCC (3 vs 1 patients), though not statistically significant. Complete and partial response rates (mRECIST criteria) were higher in the NAFLD group, while progressive disease was significantly more common in alcohol-HCC (52% vs 28%; p=0.04). Thirty-day mortality was higher in alcohol-HCC (16% vs 4%), though not statistically significant (Table 4).

NAFLD-related HCC patients demonstrated better survival outcomes than those with alcohol-related HCC. Both short-term and long-term survival were higher in the NAFLD group, with prolonged overall and progression-free survival compared with alcohol-related HCC (Table 5).

Table 1: Baseline clinical and demographic characteristics.

Parameters	NAFLD-HCC (n=25)	Alcohol-HCC (n=25)	P value
Age (in years), mean±SD	62.4±8.1	54.3±7.4	<0.01
Male sex	18 (72%)	24 (96%)	0.02
Diabetes mellitus	17 (68%)	6 (24%)	<0.01
Obesity (BMI>27 kg/m^2)	11 (44%)	3 (12%)	<0.01
AFP (ng/mL), median	80	300	0.04
Child-Pugh B/C	11 (44%)	18 (72%)	0.03
MELD score, mean±SD	12.8±3.4	17.2±4.1	<0.01
Ascites	10 (40%)	18 (72%)	0.02
Variceal bleed	3 (12%)	7 (28%)	0.1

Table 2: Radiologic tumor characteristics on CT/MRI.

Radiologic features	NAFLD-HCC (%)	Alcohol-HCC (%)	P value
Tumor size (cm)	6.1±2.4	4.2±1.9	<0.05
Solitary lesion	17 (68)	9 (36)	0.02
Multifocal lesions	7 (28)	14 (56)	0.03
Subcapsular location	10 (40)	4 (16)	0.05
Arterial enhancement	22 (88)	21 (84)	NS
Washout	18 (72)	19 (76)	NS
Enhancing capsule	15 (60)	14 (56)	NS
Fat in mass	6 (24)	3 (12)	0.27
Diffusion restriction	20 (80)	19 (76)	NS

Table 3: Background liver and portal hypertension features.

Parameters	NAFLD-HCC (%)	Alcohol-HCC (%)	P value
Nodular cirrhotic liver	14 (56)	22 (88)	<0.01
Steatosis on imaging	18 (72)	4 (16)	<0.001
Splenomegaly	11 (44)	20 (80)	<0.01
Portosystemic collaterals	10 (40)	19 (76)	<0.01
Portal vein thrombosis	4 (16)	9 (36)	0.1
Macrovascular invasion	3 (12)	7 (28)	0.15

Table 4: Treatment modalities and mRECIST response.

Parameters	NAFLD-HCC (%)	Alcohol-HCC (%)	P value
TACE received	15 (60)	18 (72)	0.36
RFA performed	3 (12)	1 (4)	0.3
Systemic therapy	7 (28)	6 (24)	NS
Complete response	3 (12)	1 (4)	0.3
Partial response	10 (40)	7 (28)	0.3
Progressive disease	7 (28)	13 (52)	0.04
30-day mortality	1 (4)	4 (16)	0.15

Table 5: Survival outcomes.

Outcomes	NAFLD-HCC	Alcohol-HCC	P value
1-year survival	68%	40%	<0.05
3-year survival	24%	8%	<0.05
Median OS (months)	18.6	10.4	<0.05
Median PFS (months)	12.3	6.8	<0.05

DISCUSSION

This study highlights distinct radiologic, clinical, and prognostic differences between NAFLD-related and alcohol-related HCC in an Indian cohort, findings that are largely consistent with observations from global literature. The demographic and clinical profiles observed reflect the divergent pathogenic mechanisms underlying these two etiologies. Patients with NAFLD-related HCC were older and exhibited significantly higher rates of diabetes and obesity, underscoring the role of metabolic syndrome-driven hepatocarcinogenesis described in earlier studies.[3,5] In contrast, alcohol-related HCC patients were younger and predominantly male, mirroring established epidemiological patterns of alcohol consumption in Asian populations.[7,20]

Lower AFP levels in the NAFLD-HCC group further distinguish this entity and have been reported previously by multiple investigators. NAFLD-related tumors frequently present with normal or only mildly elevated AFP levels, which may contribute to diagnostic delays and challenges in surveillance-based detection.[22,27]

Radiologically, NAFLD-related HCC in the present study was characterized by larger tumor size but predominantly solitary lesions, a pattern consistent with prior reports attributing delayed diagnosis to inadequate surveillance strategies, particularly in non-cirrhotic NAFLD patients.[12,18] The observation that many NAFLD-HCC cases arose in non-cirrhotic or minimally cirrhotic livers reinforces the concept of metabolic hepatocarcinogenesis

occurring independently of advanced fibrosis, as demonstrated in earlier studies.[10,11]

In contrast, alcohol-related HCC demonstrated significantly higher rates of multifocal disease and more pronounced cirrhotic morphology. These findings are in agreement with Western and Asian data linking chronic alcohol exposure to diffuse hepatic injury and aggressive, multicentric tumor development.[20,24] Features of advanced portal hypertension, including splenomegaly and portosystemic collaterals, were markedly more frequent in alcohol-HCC patients, reflecting the well-established pathophysiological consequences of alcoholic cirrhosis.[13,21]

Macrovascular invasion, including portal vein thrombosis, was also more commonly observed in alcohol-related HCC in this cohort. This aligns with previous evidence suggesting that alcohol-induced inflammation and fibrosis accelerate hepatocarcinogenesis and predispose to vascular invasion, thereby worsening disease stage at presentation.[24,25]

From a prognostic perspective, NAFLD-related HCC was associated with significantly better 1-year and 3-year survival outcomes compared with alcohol-related HCC. These findings are consistent with reports emphasizing that preserved hepatic functional reserve has a greater influence on survival than tumor size alone.[22,28] Alcohol-related HCC patients in the present study experienced more frequent decompensation events, which likely limited their tolerance to transarterial chemoembolization and systemic therapies, a phenomenon also described in earlier studies.[29]

Notably, the survival advantage observed in NAFLD-related HCC despite larger tumor size supports existing literature suggesting that metabolic HCC may exhibit less aggressive biological behavior but is often detected at a later stage due to insufficient surveillance practices.[18,23]

The clinical implications of these findings are significant. They reinforce the emerging recommendation that high-risk NAFLD patients-particularly those with diabetes, obesity, or age greater than 55 years-should be considered for regular HCC surveillance even in the absence of cirrhosis.[12,22] For alcohol-related HCC, strategies focusing on optimization of portal hypertension, early alcohol abstinence interventions, and integrated addiction-care pathways may improve eligibility for curative or disease-modifying therapies, in line with current global guidelines.[8,28]

The strengths of this study include an equal sample size across both etiological groups, allowing balanced comparison and reducing selection bias. It provides detailed radiologic characterization using standardized CT/MRI features, enabling meaningful morphologic assessment. The combined evaluation of tumor characteristics, background liver morphology, and vascular invasion offers a comprehensive imaging-based analysis. Additionally, correlation of radiologic findings with clinical parameters and survival outcomes enhances the overall interpretative value of the study.

This study has several limitations. Its retrospective, single-center design and relatively small sample size limit statistical power and generalizability.[26] Potential confounders, including prior surveillance status, alcohol exposure details, metabolic control, and treatment heterogeneity, were not fully adjusted for. Histopathologic correlation was not uniformly available, and interobserver variability in imaging assessment may have influenced findings. Larger prospective multicenter studies are needed to validate these results.

Overall, the findings of this study align closely with international data and reinforce the concept that NAFLD-related and alcohol-related HCC represent distinct clinical and radiologic entities with differing patterns of progression and survival. These results support a shift toward etiology-specific surveillance and management strategies tailored to the evolving epidemiology of HCC in India.

CONCLUSION

This study demonstrates that NAFLD-related and alcohol-related HCC represent biologically and radiologically distinct disease entities with divergent clinical outcomes in the Indian context. NAFLD-related HCC is more commonly characterized by larger, predominantly solitary tumors occurring in the setting of relatively preserved hepatic function, which translates into significantly better short-term and long-term survival despite delayed detection. In contrast, alcohol-related HCC is associated with advanced cirrhosis, multifocal tumor burden, pronounced portal hypertension, and a higher prevalence of macrovascular invasion, factors that collectively contribute to inferior prognosis.

These findings highlight important gaps in current surveillance practices, particularly among high-risk NAFLD patients who may develop HCC even in the absence of cirrhosis. At the same time, they emphasize the need for comprehensive, multidisciplinary management strategies for ALD, incorporating early abstinence interventions and optimization of liver function to improve treatment eligibility and outcomes. Overall, the results support the implementation of etiology-specific surveillance, diagnostic, and therapeutic pathways tailored to the evolving epidemiology of HCC in India, with the potential to improve early detection and survival.

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