ORIGINAL ARTICLE

Prognostic Factors and Causes of Mortality in Thai Patients with Hepatocellular Carcinoma: A Retrospective Cohort Study

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Background: Hepatocellular carcinoma (HCC) is a complex and heterogeneous disease with prognosis influenced by numerous factors but determinants and causes of mortality in Thai HCC patients remain underrepresented.

Objective: To identify prognostic factors and causes of death in Thai HCC patients at a referral hospital.

Materials and Methods: A retrospective cohort study was conducted at the Medical Oncology Unit of Sawanpracharak Hospital, Thailand, included patients diagnosed with HCC between October 2018 and September 2022. Diagnoses were confirmed by either pathological examination or characteristic image findings on multiphase computed tomography (CT) or magnetic resonance imaging (MRI). Survival data were analyzed using Kaplan-Meier and log-rank tests, followed by Cox proportional hazards regression for significant variables.

Results: Among 398 patients analyzed, 329 (82.66%) died, and the median overall survival (OS) was 4.57 months. No significant OS differences were found between viral and non-viral hepatitis-associated HCC. Independent factors for poorer survival included ECOG 3-4 (HR 1.631, p=0.007), elevated alpha fetoprotein (AFP) levels greater than 400 ng/mL (HR 1.366, p=0.031), elevated alkaline phosphatase (ALP) activity of 172 IU/L or greater (HR 2.201, p<0.001), neutrophil-to-lymphocyte ratio (NLR) of 5 or greater (HR 1.450, p=0.023), platelet-to-lymphocyte ratio (PLR) of 150 to 300 (1.423, p=0.020), Barcelona clinic liver cancer (BCLC) stage C or D (HR 3.589, p=0.001), and worse Child-Pugh (CP) scores (CP-B: HR 1.659, p=0.001; CP-C: HR 2.194, p=0.001).

Conclusion: Thai HCC patients demonstrated short survival. Advanced ECOG status, elevated AFP and ALP levels, high NLR and PLR ratios, advanced BCLC stage, and poor CP scores were associated with poor outcomes.

Keywords: Hepatocellular carcinoma; Prognosis; Survival rate

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Hepatocellular carcinoma (HCC) is a major global health challenge and the third leading cause of cancer-related death in Thailand[1]. HCC is a complex and heterogeneous disease often associated with cirrhosis and chronic viral hepatitis, which negatively impact treatment options and survival outcome[2]. Previous studies have identified several factors associated with poor outcomes, including poor performance status, advance stage as defined by the American Joint Committee on Cancer (AJCC) staging system[3] or Barcelona clinic liver cancer (BCLC) stage[3], hepatitis B virus (HBV) and hepatitis C virus (HCV) co-infection[4], elevated alpha fetoprotein (AFP) and alkaline phosphatase (ALP), low serum albumin and extrahepatic metastasis[5], and complication of cirrhosis[6]. Recent studies have highlighted the role of inflammation in tumor development[7]. Cancer-related inflammation promotes cytokine and mediator upregulation, contributing to tumor progression by inhibiting apoptosis, promoting angiogenesis, and causing DNA damage[8]. Inflammatory markers such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C-reactive protein (CRP) have been studied for their roles in tumorigenesis and prognosis across various cancers[9,10], including HCC[11]. However, most studies focused on Western populations, where environmental exposures,

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infectious risk factors, healthcare access, and HCC incidence and mortality rates differ significantly from those in Asia[12,13]. Consequently, findings from Western cohorts may not fully capture the prognostic factors and mortality causes in Thai populations. Understanding these factors is crucial for optimizing clinical decision-making and developing strategies to improve survival. The present study aimed to evaluate factors of survival and causes of death among HCC patients in Thailand, providing insights that can guide more tailored management strategies.

Materials and Methods

Study design and data collection

Prognosis research using retrospective cohort design was conducted at the medical oncology unit of Sawanpracharak Hospital, Thailand, with ethical approval from the hospital's Human Research Ethics Committee (COA no. 20/2567). Patients diagnosed with HCC (ICD-O-C22) between October 2018 and September 2022, identified through the hospital's cancer registry, which served as a tertiary care center in Thailand. HCC diagnoses were confirmed by pathology or imaging base on the American Association for the Study of Liver Diseases (AASLD) Guidelines[14] using multi-phase computed tomography (CT) or magnetic resonance imaging (MRI).

Data were extracted from electronic medical records and included demographics, clinical characteristics, hepatitis markers, laboratory results, diagnosis date, cancer stages, and treatment regimens as illustrated in the study flow diagram (Figure 1). The Child-Pugh (CP) score, calculated based on albumin, bilirubin, international normalized ratio, ascites, and encephalopathy, categorized patients into three classes, CP-A with 5 to 6 points, CP-B with 7 to 9 points, and CP-C with 10 to 15 points.

Statistical analysis

The sample size was calculated for two independent groups, assuming an 80% mortality rate and the impact of curative treatment on survival. The sample size of 278 individuals was determined after increasing by 30% to account for data loss or incompleteness. Categorical variables were analyzed using the exact probability test, while continuous variables were analyzed using either the student's t-test or the Wilcoxon rank-sum, depending on distribution. Survival time was defined as the interval from diagnosis to either death or the censoring date of December 31, 2023. Death dates were obtained from the Thailand Civil Registration database. Kaplan-Meier analysis with log-rank testing identified potential prognostic factors, and variables with p-values less than 0.05 were included in a Cox proportional-hazards regression (Cox-PH) model. All analyses were conducted using Stata Statistical Software, version 18 (StataCorp LLC, College Station, TX, USA).

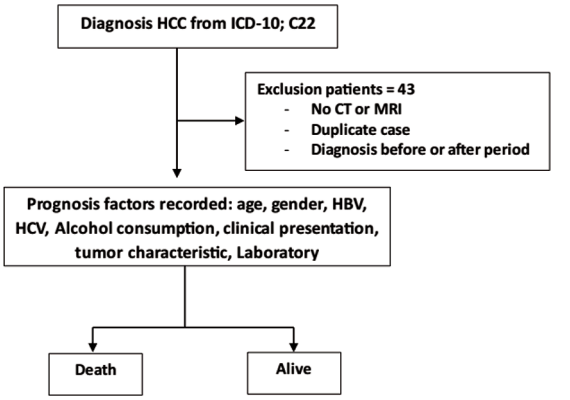

Figure 1. Study flow diagram.

Results

Demographic characteristics

Three hundred ninety-eight patients were analyzed, including 329 (82.66%) who passed away. Males predominated at 78.0%, and most patients (80.65%) had the Eastern Cooperative Oncology Group (ECOG) score of 0 to 2, which was significantly higher in the survival group. The leading underlying etiology was HBV in 219 patients (55.16%), followed by HCV in 118 patients (29.72%), non-viral causes in 47 patients (11.88%), and HBV-HCV co-infection in 13 patients (3.27%). HCC surveillance was conducted in 18.69% of cases, with a significantly higher proportion in survivors at 33.82% compared to deceased patients at 15.55% (p=0.001). The majority (59.55%) were diagnosed at an advanced stage (BCLC C-D), and only 9.05% received curative treatment. Survivors were more likely to receive curative treatment, at 30.43%, than deceased patients, at 4.56%. Patient characteristics are summarized in Table 1.

Survival and cause of death

The median overall survival (OS) was 4.57 months (95% CI 3.48 to 6.60). No significant differences in OS were observed across liver disease etiologies as non-viral at 6.47 months, HCV at 6.64 months, HBV at 4.48 months, and co-infection at

Table 1. Patient characteristic

	Total		Death		Alive		p-value
	n	Result	n	Result	n	Result	
Male; %	312	78.39	263	79.94	49	71.01	0.109
Age (year); mean±SD	398	61.0±10.43	329	61.2±10.42	69	60.0±9.95	0.541
BMI; mean±SD	378	22.5±3.86	309	22.3±3.81	69	23.4±3.97	0.032
ECOG; %							<0.001
0-2	321	80.65	253	76.90	68	98.50	
3-4	77	19.35	76	23.10	1	1.45	
Viral associated; %							0.530
Non-viral	47	11.84	37	11.28	10	14.49	
HCV	118	29.72	94	28.66	24	34.78	
HBV	219	55.16	186	56.71	33	47.33	
HBV and HCV	13	3.27	11	3.35	2	2.90	
Alcohol; %	139	35.01	121	36.89	18	26.09	0.097
Surveillance; %	74	18.69	51	15.55	23	33.82	0.001
Underlying disease; %							
HIV	7	1.76	5	1.52	2	2.90	0.350
Diabetic mellitus	73	18.39	60	18.24	13	19.12	0.864
IHD	4	1.22	4	1.22	-	-	1.000
CVD	14	3.53	12	3.66	2	2.90	1.000
Dyslipidemia	67	16.83	54	16.41	13	18.84	0.599
Child Pugh; %							<0.001
A	183	46.80	126	39.01	57	83.82	
B	138	35.29	127	39.32	11	16.18	
C	70	17.90	70	21.67	0	-	
Weight loss; %	169	42.46	156	47.42	13	18.81	<0.001
Abdominal pain; %	207	52.01	186	56.53	21	30.43	<0.001
Ascites; %	112	28.14	105	31.91	7	10.14	<0.001
Main PV; %	123	30.98	121	36.89	2	2.90	<0.001
Rupture; %	30	7.54	29	8.81	1	1.45	0.041
Multifocal liver lesion; %	263	66.08	241	73.25	22	31.88	<0.001
Lung metastasis; %	55	13.82	53	16.11	2	2.90	0.002
Peritoneal metastasis; %	8	2.01	7	2.13	1	1.45	1.000
Bone metastasis; %	15	3.77	15	4.56	0	0.00	0.084
BCLC; %							<0.001
0-A	46	11.56	19	5.78	27	39.13	
B	115	28.89	78	23.71	37	53.62	
C-D	237	59.55	232	78.52	5	7.25	
Curative treatment; %	36	9.05	15	4,356	21	30.43	<0.001
Total bilirubin (mg/dL); median (IQR)	394	1.25 (0.67 to 2.50)	325	1.35 (0.72 to 2.98)	69	0.80 (0.52 to 1.30)	<0.001
Albumin (g/dL); median (IQR)	394	3.3 (2.8 to 3.8)	325	3.20 (2.70 to 3.70)	69	3.80 (3.40 to 4.10)	<0.001
ALP (IU/L); median (IQR)	394	157 (105 to 239)	325	170 (115 to 260)	69	105 (83 to 139)	<0.001
AFP (ng/mL); median (IQR)	370	511.5 (29.89 to 7,238)	302	1,044 (63 to 12,100)	68	24 (6 to 162)	<0.001
Hb (g/dL); mean±SD	392	11.36±5.02	324	11.17±5.38	68	12.27±2.60	0.100
WBC (mm^3) median (IQR)	392	7,675 (5,310 to 9,715)	324	7,925 (5,725 to 10,155)	68	5,870 (3,880 to 7,860)	<0.001
Neutrophil (mm^3); median (IQR)	392	4,915 (3,006 to 6,975)	324	5,340 (3,323 to 7,725)	68	3,185 (2,100 to 4,700)	<0.001
Lymphocyte (mm^3); median (IQR)	392	1,440 (1,045 to 1,940)	324	1,447 (1,050 to 1,914)	68	1,425 (1,005 to 2,060)	0.761
Platelet ($\times 10^3$); median (IQR)	392	187 (122 to 283)	324	205 (134 to 292)	68	145 (85 to 199)	<0.001
INR; mean±SD	360	1.24±0.18	298	1.27±0.36	62	1.12±0.16	0.001

BMI=body mass index; ECOG=Eastern Cooperative Oncology Group; HCV=hepatitis C virus; HBV=hepatitis B virus; HIV=human immunodeficiency virus; IHD=ischemic heart disease; CVD=cardiovascular disease; PV=portal vein; BCLC=Barcelona clinic liver cancer; ALP=alkaline phosphatase; AFP=alpha fetoprotein; Hb=hemoglobin; WBC=white blood cell; INR=international normalized ratio; SD=standard deviation; IQR=interquartile range

Table 2. Cause of death and survival

	All patients (n=398)
Death; n (%)	329 (82.66)
Cause of death; n (%)	
HCC	202 (61.59)
Upper gastrointestinal bleeding	35 (10.68)
Other cirrhosis complications	9 (2.74)
Sepsis	15 (4.57)
Other cause	11 (3.35)
Unknown	65 (20.73)
OS (month); median (95% CI)	4.57 (3.48 to 6.06)
12 months survival	33.75%
24 months survival	22.99%
36 months survival	15.40%

HCC=hepatocellular carcinoma; OS=overall survival; CI=confidence interval

4.50 months (p=0.284) (Figure 2). The leading cause of death was HCC progression, followed by upper gastrointestinal bleeding in 10.68% of the cases and sepsis in 4.57% of the cases as detailed in Table 2.

Factor of HCC survival

Univariable analysis identified significant factors associated with survival, including ECOG performance status, body mass index (BMI), alcohol consumption, clinical presentation with weight loss or ascites, main portal vein (PV) involvement, tumor size exceeding 10 cm, elevated AFP levels of more than 400 ng/mL, lung or bone metastases, multifocal liver lesions, elevated ALP activity of 172 IU/L or more, NLR, and PLR (Table 3).

Multivariable analysis revealed independent predictors of mortality, including ECOG 3-4 (HR 1.631, p=0.007), AFP levels of more than 400 ng/mL (HR 1.366, p=0.031), ALP activity of 172 IU/L or more (HR 2.201, p<0.001), NLR of 5 or more (HR 1.450, p=0.023), PLR of 150 to 300 (1.423, p=0.020), BCLC stage C or D (HR 3.589, p=0.001), CP-B (HR 1.659, p=0.001), and CP-C (HR 2.194, p=0.001). Detailed results are presented in Table 3 and Figure 3.

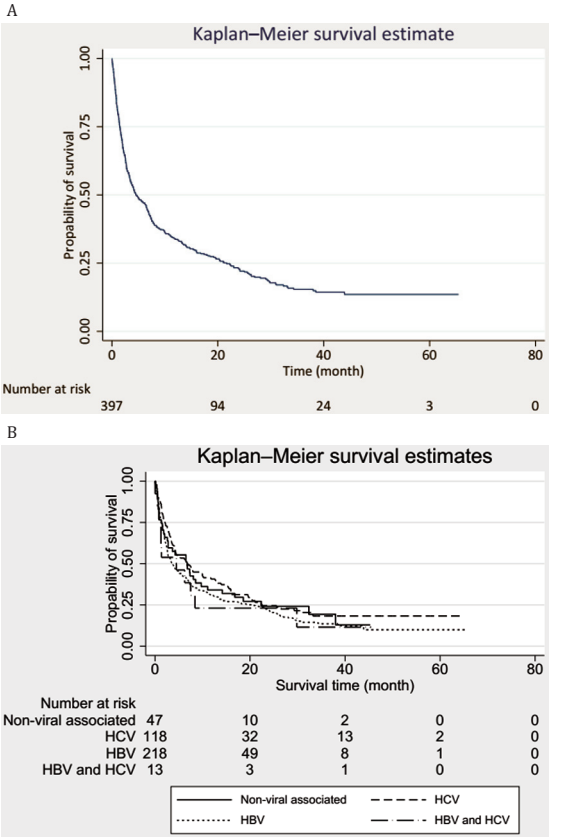

Figure 2. Kaplan-Meier survival. (A) Overall survival, (B) Survival according to viral associated HCC.

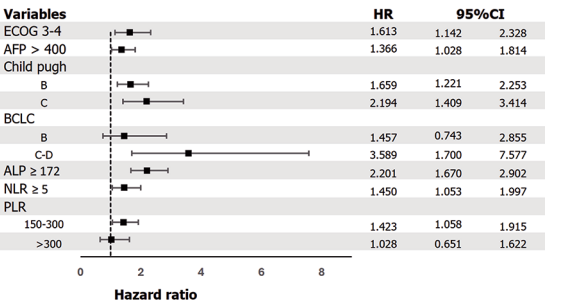

Figure 3. Forest plot of multivariable Cox regression model illustrating the prognostic factors predict OS HCC.

Discussion

The present study identified median OS of 4.56 months and a 12-month OS rate of 33.75% lower than reported in Western populations[12,13,15]. Several factors may explain this discrepancy. First, 59.55% of patients were diagnosed at advanced stage, BCLC stages C-D, limiting eligibility for curative therapies such as surgery or radiofrequency ablation to just 10%. Second, limited access to advanced systemic therapies, including targeted agents and immunotherapies, have been shown to improve survival in advanced-stage HCC impact outcomes[16-18]. Additionally, the low HCC screening rate, at 18.69%, contributed to missed opportunities for early detection. However, trends indicate increasing HCC screening from 5.4% in 2003 to 2006 to 8.8% in 2011 to 2013[15]. Ultrasonography and AFP screening in high-risk populations have demonstrated

Table 3. Univariable and multivariable cox-regression for HCC death

Variable	n	mOS (month)	Univariable analysis			Multivariable analysis		
			HR	95% CI	p-value	HR	95% CI	p-value
ECOG								
0-2	321	7.20	Ref.			Ref.		
3-4	77	1.25	3.496	2.672 to 4.573	<0.001	1.631	1.142 to 2.329	0.007
Alcohol use	139	3.49	1.323	1.05 to 1.658	0.015	1.150	0.887 to 1.493	0.291
Viral associated HCC								
Non-viral	47	6.74	Ref.					
HCV	118	6.64	0.899	0.61 to 1.316	0.586			
HBV	219	3.48	1.137	0.799 to 1.619	0.474			
HBV and HCV	13	4.50	1.223	0.624 to 2.399	0.557			
BMI								
≥25	82	10.82	Ref.			Ref.		
<25	296	4.04	1.361	1.030 to 1.799	0.003	1.360	0.990 to 1.867	0.057
Weight loss								
No	228	9.96	Ref.			Ref.		
Yes	169	2.56	2.00	1.605 to 2.498	<0.001	1.088	0.765 to 1.320	0.968
Ascites								
No	285	7.36	Ref.			Ref.		
Yes	112	1.77	2.391	1.885 to 3.03	<0.001	1.133	0.799 to 1.480	0.590
Main PV involved								
No	273	8.81	Ref.			Ref.		
Yes	123	1.94	3.155	2.485 to 4.005	<0.001	1.158	0.850 to 1.578	0.351
Rupture								
No	368	5.03	Ref.			Ref.		
Yes	30	1.94	1.818	1.233 to 2.682	0.003	0.948	0.545 to 1.648	0.850
Tumor size								
≤10 cm	279	8.46	Ref.			Ref.		
>10 cm	118	2.10	2.480	1.961 to 3.138	<0.001	1.195	0.882 to 1.619	0.249
Multifocal liver lesion								
No	135	22.29	Ref.			Ref.		
Yes	262	2.70	2.748	2.139 to 3.531	<0.001	1.0040	0.7525 to 1.391	0.976
Lung metastasis								
No	342	6.51	Ref.			Ref.		
Yes	55	1.74	2.456	1.821 to 3.313	<0.001	1.167	0.815 to 1.672	0.398
Bone metastasis								
No	382	4.86	Ref.			Ref.		
Yes	15	2.71	1.923	1.142 to 3.237	0.014	1.461	0.785 to 2.717	0.231
AFP								
≤400	177	13.91	Ref.			Ref.		
>400	193	2.50	2.424	1.920 to 3.062	<0.001	1.366	1.028 to 1.814	0.031
Child Pugh								
A	183	15.55	Ref.			Ref.		
B	138	2.69	2.695	2.095 to 3.468	<0.001	1.659	1.221 to 2.253	0.001
C	70	1.15	5.851	4.293 to 7.973	<0.001	2.194	1.409 to 3.414	0.001
BCLC								
0-A	46	38.50	Ref.			Ref.		
B	115	15.35	2.156	1.304 to 3.564	0.003	1.457	0.743 to 2.855	0.273
C-D	237	20.01	9.043	5.598 to 14.607	<0.001	3.589	1.700 to 7.577	0.001

mOS=median overall survival; HR=hazard ratio; CI=confidence interval; ECOG=Eastern Cooperative Oncology Group; HCC=hepatocellular carcinoma; HBV=hepatitis B virus; HCV=hepatitis C virus; BMI=body mass index; PV=portal vein; AFP=alpha fetoprotein; ALP=alkaline phosphatase; BCLC=Barcelona clinic liver cancer; Hb=hemoglobin; NLR=neutrophil-to-lymphocyte ratio; PLR=platelet-to-lymphocyte ratio

Table 3. (continued)

Variable	n	mOS (month)	Univariable analysis			Multivariable analysis		
			HR	95% CI	p-value	HR	95% CI	p-value
Curative treatment								
Yes	36	38.49	Ref.			Ref.		
No	361	3.68	4.595	2.726 to 7.744	<0.001	1.654	0.800 to 3.5420	0.174
ALP								
<172	223	13.56	Ref.			Ref.		
≥172	117	2.17	2.823	2.245 to 3.550	<0.001	2.201	1.670 to 2.902	<0.001
Hb								
>13	75	6.93	Ref.			Ref.		
≤13	316	4.24	1.470	1.094 to 1.977	0.011	1.352	0.953 to 1.918	0.090
NLR								
<5	274	7.49	Ref.			Ref.		
≥5	117	2.04	2.3133	1.829 to 2.927	<0.001	1.450	1.053 to 1.997	0.023
PLR								
<150	215	8.42	Ref.			Ref.		
150 to 300	130	2.56	1.810	1.42 to 2.296	<0.001	1.423	1.058 to 1.915	0.020
>300	46	2.56	1.921	1.361 to 2.713	<0.001	1.028	0.651 to 1.622	0.905

mOS=median overall survival; HR=hazard ratio; CI=confidence interval; ECOG=Eastern Cooperative Oncology Group; HCC=hepatocellular carcinoma; HBV=hepatitis B virus; HCV=hepatitis C virus; BMI=body mass index; PV=portal vein; AFP=alpha fetoprotein; ALP=alkaline phosphatase; BCLC=Barcelona clinic liver cancer; Hb=hemoglobin; NLR=neutrophil-to-lymphocyte ratio; PLR=platelet-to-lymphocyte ratio

improved early detection[15,19,20], increased curative treatment rates, and 37% reduction in HCC mortality in chronic hepatitis patient[21]. Emerging biomarkers such as protein induced by vitamin K absence of antagonist-II (PIVKA-II) and AFP-L3 show promising for future clinical applications[22]. HBV was the common underlying etiology, followed by HCV reflecting regional differences due to lack of universal HBV vaccination program before 1992 and has insufficiency HCV screening in Thailand. The lack of significant survival differences between various etiologies such as HBV, HCV, and non-viral causes, is in contrast to findings from Western studies, where non-viral associated HCC typically correlated with poorer outcomes[23,24]. In Western populations, HCC is primarily non-viral, and routine screening among HBV and HCV carriers, which facilitates early detection[13,15]. In contrast, the present study found a predominance of viral-associated HCC and low screening rates, resulting in most patients being diagnosed at an advanced stage. This contributed to the absence of significant differences in survival outcomes. HCC progression, for 77.10%, was a leading cause of death, which is consistent with the aggressive nature of the disease, particularly in patients diagnosed at advanced stage. Gastrointestinal bleeding, for 9.54%, was the second most common cause of death. This reflects the complications of cirrhosis. These findings emphasize the need to improve care pathways and proactive management strategies for chronic liver disease.

The present study identified independent prognostic factors for HCC survival, including ECOG performance status 3-4, elevated AFP level, elevated ALP activity, NLR of 5 or greater, PLR between 150 and 300, BCLC stage C-D, and CP class B or C. Previous studies have demonstrated that CP score, a marker of liver function, is a key prognostic factor in cirrhosis patients[25]. As CP is integrated into the BCLC staging system, the two variables are inherently correlated. The BCLC stage, which guides treatment selection, places patients with CP-C in stage D, excluding them from therapies like trans arterial chemoembolization (TACE) or systemic treatments. Therefore, relying solely on CP score to predict HCC outcomes may introduce bias.

Elevated ALP, a marker of biliary disease, was identified as a prognostic factor for HCC, aligning with Su et al. (2022)[26], where ALP of 172 IU/L or greater was associated with shorter survival of 7.7 versus 55.4 months (HR 1.594, 95% CI 1.377 to 1.820, p<0.001). ALP has also been linked to HCC recurrence after hepatectomy[27,28]. AFP is widely used for screening HCC in cirrhotic patients and considered as a diagnostic marker[29]. It also serves as prognostic marker. Studies demonstrated that AFP of 400 ng/mL or greater correlated with larger tumor, vascular invasion, poor differentiation,

and lower overall and disease-free survival post hepatectomy[30,31], and worse outcomes with sorafenib treatment[16,32]. These finding, along with the present study's result, suggest that AFP's prognostic value, though further research is needed to determine the optimal cut-off.

Inflammatory markers such as NLR and PLR are established prognostic markers in various cancers[9-11,33,34]. The role of proinflammatory cytokine and growth factors, released by tumors and their microenvironment, in tumor development has been well-document[7]. Neutrophils suppress the cytokine activity of immune cells including lymphocytes, activated T-cell and natural killer cell, thereby promoting tumor progression[35,36]. Conversely, high tumor-infiltrating lymphocyte level improves responses to cytotoxic therapies[37]. Elevated NLR correlated with peritumor macrophages infiltration and increased interleukin (IL)-17 levels[38]. While PLR reflect platelet-driven tumor angiogenesis[39], both are suggested to be crucial determinants tumor growth. Both markers are accessible and show promise as prognostic markers in HCC, through optimal clinical cutoffs require further research.

ECOG performance status, reflecting a patient's general health and ability to perform daily activities, significantly impacts cancer prognosis and treatment decisions[40,41]. It is a key variable of BCLC staging system. Although the BCLC system has shown a concordance index, C-statistics, ranging from 0.54 to 0.8 and is effective for prognosticating patients with cirrhosis and curative HCC, it is less applicable to advanced HCC cases undergoing systemic therapy[42]. The BCLC system recommends systemic or TACE for patients with an ECOG 0-1, while those with higher ECOG score typically received best supportive care. Emerging evidence suggests that HCC patients with ECOG of 1 or greater may benefit from more active treatment[43-45]. In Thailand, where most HCC cases present at advanced stage (BCLC B-C), the BCLC system alone may be insufficient for prognostic stratifying in systemic therapies candidates.

The present study has limitations. First, its retrospective design restricted analysis to available medical records. Second, it was conducted in Thailand, where the government universal health coverage limited access to novel HCC treatments, potentially affecting survival outcomes.

Conclusion

The present study highlights the poor OS of HCC patients, given the high prevalence of advanced disease. Impaired liver function, and elevated inflammatory markers, poor ECOG, and high stage BCLC were independent mortality predictors. The findings emphasize the importance of early diagnosis, enhance HCC screening and improved management of underlying liver diseases is essential to improve the outcomes. Additionally, biomarkers such as AFP, ALP, NLR, and PLR may serve as valuable prognostic tools.

What is already known about this topic?

HCC is the leading cause of cancer-related mortality in Thailand. Key factors influencing HCC survival include tumor burden, ECOG performance status, and cirrhosis-related complications.

What does this study add?

The 12-month OS rate for patients with HCC is 33.75% with HCC being the leading cause of death followed by upper gastrointestinal bleeding. This study revealed low HCC surveillance, leading to missed opportunities for early detection and contributing to poorer survival outcomes compared to Western populations. Notably, over half of HCC patients are diagnosed at advanced stages. Key predictors of poor prognosis include ECOG 3-4, AFP levels of 400 ng/mL or greater, ALP of 172 IU/L or greater, NLR of 5 or greater, PLR between 150 and 300, BCLC stage C-D, and CP class B-C. These findings underscore the need to enhance HCC screening programs for high-risk populations and inform clinical decision-making to improve patient outcome.

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Data availability

The data that support the findings of the present study are available upon request from the corresponding author. The data is not publicly available due to privacy or ethical restrictions.

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Conflicts of interest

The author declares no conflicts of interest.

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