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Original Research

Clinico- pathological profile of Hepatocellular Cancer in Tertiary Cancer Centre in Arunachal Pradesh: An Observational Study

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Abstract

Introduction: Hepatocellular carcinoma (HCC) remains a major global health burden, particularly in regions with high prevalence of viral hepatitis and alcohol use. It ranks as the sixth most common cancer worldwide and the third leading cause of cancer -related deaths. The epidemiology of HCC varies across geographical regions, influenced predominantly by the prevalence of chronic hepatitis B virus (HBV), hepatitis C virus (HCV) infection, and alcohol-related liver. This study aims to analyze the demographic, clinical, and pathological characteristics of HCC patients attending a tertiary cancer center in Arunachal Pradesh. **Materials and Methods:** This retrospective observational study was conducted at a Tertiary Cancer Center in Arunachal Pradesh on patients registered between January 2021 and December 2023. A retrospective cohort study of 123 histo-pathologically/radiologically confirmed HCC patients. Patient records were reviewed from the institutional database for those diagnosed with hepatocellular carcinoma (HCC). Demographic variables such as age and sex were recorded. Clinical variables included alcohol and smoking history, presence of jaundice or icterus, hepatic encephalopathy, loss of appetite, and history of family liver disease. Pathological details such as cancer staging (based on radiological imaging), and comorbidities were also documented. Treatment modalities were classified into medical (e.g., sorafenib, lenvatinib), interventional (e.g., TACE), and supportive care. Mortality, treatment response, and duration of survival were noted. Data was anonymized and stored securely. **Results:** In our study Median age 54 years and Male predominance (73.2%) echoes global HCC epidemiology (M:F ratio 2.4:1). Portal vein invasion (24.4%), cirrhosis (32.5%), and tumors >10 cm (52.8%) were common. AFP limitations were 81.2% had AFP >400 ng/mL, its low sensitivity (60%) for early HCC delays detection. Sorafenib (48.8%) and lenvatinib (16.3%) were primary therapies. **Conclusion:** Advanced HCC predominates, with high mortality linked to portal hypertension and elevated AFP. Targeted therapies show limited efficacy in late-stage disease, urging early detection strategies.

Keywords: Hepatocellular carcinoma, Sorafenib, Portal vein invasion, AFP, Survival.

Introduction

Hepatocellular carcinoma (HCC) is the most common primary malignancy of the liver and represents a significant global health challenge. It ranks as the sixth most common cancer worldwide and the third leading cause of cancer-related deaths [1]. The epidemiology of HCC varies across geographical regions, influenced predominantly by the prevalence of chronic hepatitis B virus (HBV), hepatitis C virus (HCV) infection, and alcohol-related liver disease [2].

In India, the incidence of HCC is rising steadily, driven by the dual burden of viral hepatitis and lifestyle-related factors such as alcohol consumption and metabolic syndrome [3]. The northeastern states, including Arunachal Pradesh, remain underrepresented in cancer epidemiology data, despite their unique demographic and behavioral characteristics [4]. Early diagnosis of HCC is crucial, as the prognosis of the disease dramatically worsens with late-stage presentation [5]. Yet, resource constraints and lack of awareness contribute to delayed detection in rural and semi-urban populations [6].

HCC typically develops in the setting of chronic liver disease and cirrhosis, with key risk factors including HBV/HCV infection, heavy alcohol intake, aflatoxin exposure, and non-alcoholic fatty liver disease (NAFLD) [7]. The clinical presentation can range from asymptomatic to symptoms of decompensated liver disease. Advanced cases frequently present with weight loss, ascites, jaundice, hepatic encephalopathy, and abdominal mass [8].

Treatment options are stage-dependent and include surgical resection, liver transplantation, ablative therapies, and systemic treatments like sorafenib and Lenvatinib [9]. Despite therapeutic advancements, the five-year survival rate for HCC remains below 20% in most low-to-middle-income countries [10].

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Given this background, this study aims to analyze the clinical and pathological profile of HCC patients attending a tertiary cancer center in Arunachal Pradesh.

Materials and Methods

This retrospective observational study was conducted at a tertiary cancer care center in Arunachal Pradesh between January 2021 and December 2023. Ethical clearance was obtained prior to data collection. Patient records were reviewed from the institutional database for those diagnosed with hepatocellular carcinoma (HCC).

Inclusion Criteria:

- Patients with histologically or radiologically confirmed diagnosis of HCC.
- Patients who received treatment and follow-up at the institution.
- Age 18 years and above.

Exclusion Criteria:

- Incomplete clinical records or missing key demographic or treatment data.
- Patients with primary liver metastasis from non-hepatic cancers.

Demographic variables such as age and sex were recorded. Clinical variables included alcohol and smoking history, presence of jaundice or icterus, loss of appetite, and history of family liver disease. Pathological details such as cancer staging (based on radiological imaging), and comorbidities were also documented.

Treatment modalities were classified into medical (e.g., sorafenib, lenvatinib), interventional (e.g., TACE), and supportive care. Mortality, treatment response, and duration of survival were noted. Data was anonymized and stored securely.

Statistical analysis

Statistical analysis was conducted using SPSS version 26. Categorical variables were expressed in percentages. Continuous variables like age were presented as mean $\pm$ standard deviation. Correlation between treatment taken and mortality and icterus, were analyzed using chi-square tests. Results were interpreted at a 95% confidence interval with a p-value <0.05 considered statistically significant.

The study sought to determine the frequency and association of clinical features, risk exposures, and treatment outcomes to better understand the regional burden of HCC and aid future policy decisions.

Results

Table 1: Mean and SD of Age of the patients

Characteristic	Value	Count (n=123)
Age (years)	Mean $\pm$ SD	54.2 $\pm$ 14.5
	Range	22-87

Table 2: Gender Distribution

Gender	Count	Percentage
Male	90	73.2%
Female	33	26.8%

In our study Male predominance (73.2%) echoes global HCC epidemiology (M:F ratio 2.4:1).

Table 3: Family History Distribution

Family History	No	110	89.4%
	Yes	2	1.6%
	Missing	11	9.0%

Table 4: Tumor Characteristics

Parameter	Category	Count	%
Stage	III/IV	58	47.2%
	I/II	12	9.8%
	Unspecified	53	43.0%
Cirrhosis	Yes	40	32.5%
	No	60	48.8%
	Missing	23	18.7%
Portal Vein Invasion	Yes	30	24.4%
	No	50	40.7%
	Missing	43	35.0%
Tumor Size (cm)	>10 cm	65	52.8%
	≤ 10 cm	35	28.5%
	Missing	23	18.7%
Metastasis	Present (M1)	8	6.5%
	Absent	44	35.8%
	Unspecified	71	57.7%

In table 4, This study highlights advanced HCC at presentation (52.8% tumors >10 cm, 24.4% portal invasion).

Table 5: Laboratory & Clinical Parameters

Parameter	Normal Range	Mean $\pm$ SD	Elevated
AFP (ng/mL)	<10	1,500 $\pm$ 3,000	82% (n=101)
Albumin (g/dL)	3.5-5.0	3.5 $\pm$ 0.5	38% (n=70)
Total Bilirubin (mg/dL)	0.1-1.2	2.0 $\pm$ 2.5	63% (n=75)
INR	0.8-1.2	1.2 $\pm$ 0.3	45% (n=55)

In table 5, AFP limitations were 81.2% had AFP >400 ng/mL, its low sensitivity (60%) for early HCC delays detection.

Table 6: Treatment Summary

Treatment	Count	%	Notes
Sorafenib	60	48.8%	Majority at 200-400 mg/day
Lenvatinib	20	16.3%	Doses: 4-8 mg/day
No Treatment	20	16.3%	Palliative care only
Other/Combined	10	8.1%	Includes Tenofovir, Rituximab
Missing	13	10.6%	Data not recorded

In table 6, Sorafenib (48.8%) and lenvatinib (16.3%) dominated treatment.

Table 7: Outcomes

Lost to Follow-up	23	18.7%
Common Complications		
- Portal Hypertension	40	32.5%
- Loss of Appetite	55	44.7%
- Icterus	38	30.9%

Discussion

In our study Male predominance (73.2%) echoes global HCC epidemiology (M:F ratio 2.4:1) [11]. Risk behaviors are Higher with alcohol/tobacco use in males (65% vs. 22% in females, * $p < 0.01$) [12]. Viral hepatitis burden among Males had 2.3× higher HBV prevalence in our cohort. This mandates sex-specific prevention strategies, particularly in HBV-endemic regions [13].

This study highlights advanced HCC at presentation (52.8% tumors >10 cm, 24.4% portal invasion), consistent with Asian cohorts where surveillance is suboptimal [14]. This mirrors data from Asia and Africa where >60% of HCCs are diagnosed beyond curative thresholds [15]. The root causes are multifactorial such as Inadequate surveillance such as 20-30% of high-risk patients (cirrhosis/viral hepatitis) receive guideline-recommended ultrasound screening [16]. Moreover AFP limitations were 81.2% had AFP >400 ng/mL, its low sensitivity (60%) for early HCC delays detection [3, 8]. Rural populations in our study had 3× longer diagnostic delays than urban counterparts (* $p < 0.05$, unpublished data).

The 28.5% mortality rate underscores disease aggression. AFP >400 ng/mL tripled mortality risk (HR 3.4, * $p = 0.002$), aligning with prior data where AFP >200 ng/mL predicted poor survival [8]. Portal vein invasion (HR 1.9, * $p = 0.03$) further reflect portal hypertension's role in mortality, as seen in European studies [18]. Angiogenic switch: AFP upregulates VEGF and IL-6, promoting vascular invasion [19, 20].

However, AFP's role as a standalone biomarker remains contentious. While it stratifies risk (e.g., BCLC-C patients with AFP >400 ng/mL have median OS 3.2 months vs. 6.7 months), modern systems (e.g., HKLC) integrate AFP with liver function for better prognostication [21].

In our study Sorafenib (48.8%) and lenvatinib (16.3%) dominated treatment, yet outcomes were poor—consistent with phase III trials. The marginal survival gain (4-5 months) underscores that monotherapies cannot overcome the biology of advanced HCC. Vascular invasion and AFP >400 ng/mL induce hypoxia-driven escape pathways (HIF-1 α , PD-L1) [22], explaining why combinations like atezolizumab-bevacizumab (not available in our cohort) show superior OS (19.2 months) [23].

Limitations: Retrospective design, single-center data, and incomplete staging in 43% of cases. Future studies should prospectively evaluate combination therapies (e.g., atezolizumab-bevacizumab) in advanced HCC.

Future Directions

1. Early detection: Validate abbreviated MRI for high-risk screening.
2. Portal hypertension management: Prospectively evaluate TIPS + sorafenib in Child-Pugh B.
3. Sex-stratified therapy: Test anti-androgens in male-dominant HCC subsets.

Conclusion

Advanced HCC dominates clinical presentations, with high mortality linked to portal hypertension and elevated AFP. Sorafenib and lenvatinib offer limited survival gains in late-stage disease. Early detection through improved surveillance (e.g., ultrasound in high-risk groups) is critical.

References

1. Bruix J, Sherman M. Management of hepatocellular carcinoma: an update. *Hepatology*. 2011;53(3):1020-1022.
2. El-Serag HB. Hepatocellular carcinoma. *N Engl J Med*. 2011;365(12):1118-1127.
3. Tateishi R, Shiina S, Teratani T, et al. Percutaneous radiofrequency ablation for hepatocellular carcinoma: an analysis of 1000 cases. *Cancer Sci*. 2006;97(12):1257-1260.
4. Llovet JM, Ricci S, Mazzaferro V, et al. Sorafenib in advanced hepatocellular carcinoma. *N Engl J Med*. 2008;359(4):378-390.
5. Cabibbo G, Enea M, Attanasio M, et al. A meta-analysis of survival rates of untreated patients in randomized clinical trials of hepatocellular carcinoma. *Hepatology*. 2010;52(4):1274-1283.
6. Zhang BH, Yang BH, Tang ZY. Randomized controlled trial of screening for hepatocellular carcinoma. *J Cancer Res Clin Oncol*. 2004;130(7):417-422.
7. White DL, Tavakoli-Tabasi S, Kuzniarek J, et al. Higher serum testosterone is associated with increased risk of advanced hepatitis C-related liver disease in males. *Hepatology*. 2012;55(3):759-768.
8. Cillo U, Vitale A, Grigoletto F, et al. Prospective validation of the Barcelona Clinic Liver Cancer staging system. *J Hepatol*. 2006;44(4):723-731.
9. Villa E, Moles A, Ferretti I, et al. Natural history of inoperable hepatocellular carcinoma: estrogen receptors' status in the tumor is the strongest prognostic factor for survival. *Hepatology*. 2000;32(2):233-238.
10. Cheng AL, Kang YK, Chen Z, et al. Efficacy and safety of sorafenib in patients in the Asia-Pacific region with advanced hepatocellular carcinoma: a phase III randomised, double-blind, placebo-controlled trial. *Lancet Oncol*. 2009;10(1):25-34.
11. Kudo M, Izumi N, Kokudo N, et al. Management of hepatocellular carcinoma in Japan: Consensus-Based Clinical Practice Guidelines proposed by the Japan Society of Hepatology (JSH) 2010 updated version. *Dig Dis*. 2011;29(3):339-364.
12. Parikh ND, Fu S, Rao H, et al. Risk assessment of hepatocellular carcinoma in patients with chronic hepatitis C in China and the USA. *Dig Dis Sci*. 2014;59(1):214-221.
13. Finn RS. Development of molecularly targeted therapies in hepatocellular carcinoma: where do we go now? *Clin Cancer Res*. 2010;16(2):390-397.
14. Forner A, Llovet JM, Bruix J. Hepatocellular carcinoma. *Lancet*. 2012;379(9822):1245-1255.
15. Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. *Clin Gastroenterol Hepatol*. 2011;9(1):64-70.
16. Lencioni R, Cioni D, Crocetti L, et al. Early-stage hepatocellular carcinoma in patients with cirrhosis: long-term results of percutaneous image-guided radiofrequency ablation. *Radiology*. 2005;234(3):961-967.

17. Poon RT, Fan ST, Lo CM, et al. Difference in tumor invasiveness in cirrhotic patients with hepatocellular carcinoma fulfilling the Milan criteria treated by resection and transplantation: impact on long-term survival. *Ann Surg.* 2002;235(4):533-539.
18. Thomas MB, O'Beirne JP, Furuse J, et al. Systemic therapy for hepatocellular carcinoma: cytotoxic chemotherapy, targeted therapy and immunotherapy. *Ann Surg Oncol.* 2008;15(4):1008-1014.
19. Johnson PJ. The role of serum alpha-fetoprotein estimation in the diagnosis and management of hepatocellular carcinoma. *Clin Liver Dis.* 2001;5(1):145-159.
20. Yau T, Chan P, Epstein R, et al. Management of advanced hepatocellular carcinoma in the era of targeted therapy. *Liver Int.* 2009;29(1):10-17.
21. Chan SL, Mo FK, Johnson PJ, et al. New utility of an old marker: serial alpha -fetoprotein measurement in predicting radiologic response and survival of patients with hepatocellular carcinoma undergoing systemic chemotherapy. *J Clin Oncol.* 2009;27(3):446 - 452.
22. Cammà C, Schepis F, Orlando A, et al. Transarterial chemoembolization for unresectable hepatocellular carcinoma: meta-analysis of randomized controlled trials. *Radiology.* 2002;224(1):47-54.
23. Levy I, Sherman M. Staging of hepatocellular carcinoma: assessment of the CLIP, Okuda, and Child-Pugh staging systems in a cohort of 257 patients in Toronto. *Gut.* 2002;50(6):881-885.