International Journal of Scientific Reports
Mukherjee S et al. Int J Sci Rep. 2015 May;1(1):69-73
http://www.sci-rep.com
pISSN 2454-2156 | eISSN 2454-2164

DOI: http://dx.doi.org/10.18203/issn.2454-2156.IntJSciRep20150205

Research Article

Hepatocellular carcinoma in eastern India, a detail analytical report from a tertiary care hospital

Sudeb Mukherjee[1]*, Kingshuk Dhar[2], Suhana Datta[1], Apurba Kumar Mukherjee[1]

[1]Department of General Medicine, R.G. Kar Medical College, Kolkata-700004, West Bengal, India
[2]Department of Gastroenterology, R.G. Kar Medical College, Kolkata-700004, West Bengal, India

Received: 14 April 2015
Accepted: 18 April 2015

***Correspondence:**
Dr. Sudeb Mukherjee,
E-mail: drsumukherjee@gmail.com

ABSTRACT

Background: Hepatocellular carcinoma is the one of the commonest tumour worldwide. A detailed clinical profile including its etiology and vast presentation is not available in Eastern India.
Methods: Retrospective chart review of 90 patients with HCC was done. Total 90 patients (male 81, female 9) fulfilling diagnostic criteria for HCC adopted by Barcelona-2000 EASL conference were analyzed for clinical, etiological ,biochemical and radiological profile.
Results: Underlying cirrhosis was seen in 60% cases with Hepatitis B virus being the most common (33.3%) etiologic agent followed by Alcoholism (26.6%) in cirrhotic range. In 76.7% of HCC patients have AFP level more than 500 unit and practically diagnostic of HCC. Almost all patients presented with advanced disease (96.7%). Only 3.3% of HCC patients presented with mild disease.
Conclusions: The characteristics of HCC in eastern India are somewhat different from the rest of worlds. Alcohol and HBV infection are the two most important etiology prevailing here.

Keywords: HCC (Hepatocellular carcinoma), AFP (Alpha fetoprotein), HBV (Hepatitis B virus), HCV (Hepatitis C virus)

INTRODUCTION

Hepatocellular carcinoma is the commonest primary malignant tumor of the liver. It is the fifth most common cancer in men and the eighth most common in women, and it ranks fourth in annual cancer mortality rates.[1,2]

The annual global incidence is approximately 1 million cases, with male preponderance. The incidence rates varies from low-incident countries (like US) to intermediate (like Austria, South Africa) to high areas in the orient (China and Korea). In India, the mean incidence of HCC in four population-based registries is 2.77% for males and 1.38% for females. The prevalence of HCC in India varies from 0.2% to 1.6%.[3,4] However, there is paucity of published literature on profile of HCC patients in eastern India. Therefore we studied these patients for detail analysis of their demographic characteristics.

METHODS

It is a cross sectional, observational, hospital based study. Total 90 HCC patients who were admitted in department of medicine and gastroenterology, R.G.K.M.C.H, Kolkata, from 2007-2013 were retrospectively analyzed. The diagnosis of HCC was based on EASL diagnostic criterias.[5] All clinical, biochemical, serological, radiological and endoscopical details were noted from the case records and analysed accordingly. Other space occupying lesions in liver like metastasis or primary benign tumors were excluded from the study.

RESULTS

Demographic: Out of the 90 (n=90) HCC patients 81 are male (90%), only 9 patients are female (10%). There is definite male preponderance. Male:Female = 81:9 = 9:1. Out of 90 (n=90) patients 75 are Hindu (83.4%) & 15 are Muslim (16.6%). Maximum patients are in the age group of 40-59 years. That is, HCC is occurring maximally in the middle age (43.3%). Next common age group is above 60 years (33.4%).

Clinical: (clinical profile shown in Table 1 & 2) most common symptom is anorexia (96.6%). But it is a nonspecific one. Other nonspecific symptoms are weight loss (83.3%), swelling of face (26.6%). Among gastrointestinal tract related symptoms most common is pain abdomen (80%) and then abdominal swelling (66.6%), jaundice (60%). Impaired consciousness due to metastasis to brain or due to hepatic failure present in 13.3% of patients. 83.3% have poor nutrition, due to cancer cachexia or anorexia. 53% have hepatic facies, predominantly due to previous chronic liver disease. 70% have pallor, due to poor nutrition, hematemasis and/or melaea. 60% have jaundice, quite a common feature. Only 26.6% have clubbing and edema, not a common feature. Lymphadenopathy is not a feature of HCC. Hypotension is due to poor general condition. Hypertension is most probably part of nonalcoholic fatty liver disease. Respiratory distress may be due to pleural effusion - may be due to metastasis. Hepatomegaly is present in all patients (100%). 43.3% have splenomegaly and it is mostly due to portal hypertension. Ascites present in 73.3% of patients. It may be due to chronic liver disease or metastases. In 66.6% cases liver enlarged more than 5 cm and mostly hard and nodular (96.6%). 80% livers are tender. In 73.3% cases Rt lobe enlargement is more than left lobe. In 20% cases hepatic bruit are present.

Table 1: Showing distributions of clinical profile (symptoms) of all patients (n=90).

Symptoms	Number	%(n=90)
Fever	15	16.6
Weight loss	75 (5)	83.3 (16.6)
Anorexia	87	96.6
Epistaxis	3	3.3
Headache	3	3.3
Swelling of feet	24	26.6
Swelling of whole body	6	6.6
Respiratory distress	6	6.6
Pain abdomen	72	80
Vomiting	3	3.3
Abdominal swelling	60	66.6
Jaundice	54	60
Blood vomiting	3	3.3
Black tarry stool	15	16.6
Total	426	473

Most commonly involved system other than gastrointestinal system is respiratory system (6%) in the form of pleural effusion. Lung parenchymal metastasis is not present in our study population. Pleural effusion may be due to chronic liver disease or pleural metastasis. Central nervous system involvement occurred in the form of impaired consciousness - may be due to hepatic failure or brain metastasis (Table 1 & 2).

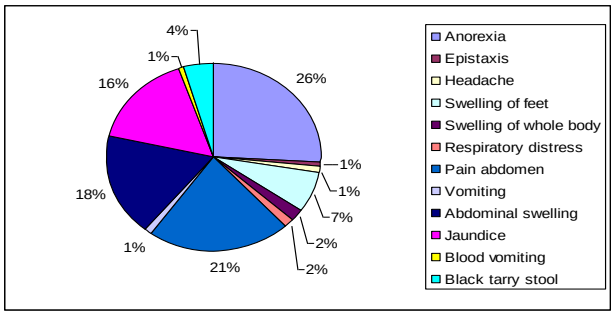

Figure 1: Showing distribution of symptoms in all patients (n=90).

Table 2: Distributions of clinical profile (signs on general examination) of all patients (n=90).

Signs	Number	%(n=90)
Impaired conscious	12	13.3
Poor nutrition	75	83.3
Hepatic facies	48	53.3
Pallor	63	70
Jaundice	54	60
Clubbing	24	26.6
Oedema	24	26.6
Enlarged lymph node	0	0
Hypertension	6	6.6
Hypotension	6	6.6
Abnormal pulse	0	0
Increased respiratory rate	6	6.6
Total	348	353

Etiological: 56.5% HCC patients are alcoholic - 26.6% had ingested alcohol in the cirrhotic risk range & 23.3% had ingested in the fatty liver risk range. 56.6% of patients are addicted to alcohol, 70% of them are addicted to tobacco (including one patient who is addicted to gurakhu). 46.4 % of them are addicted to both tobacco and alcohol. Only 20% of them have no addiction. Majority of patients have no significant past history related to gastrointestinal tract. 40% give definite past history of chronic liver disease which is a predisposing factor for HCC. 30% give history of fatty liver which was detected by ultrasonography and it is associated with alcohol intake. Only 16.6% give history of jaundice but the cause was not known to them. Only 16.6% of HCC patients have other risk factors. Even if they are predisposing factors for HCC, they are not prevalent at all. 33.3% of HCC patients are HbsAg +ve. 3.3% has Ab against HCV (Table 3 & 4).

Table 3: Distributions of all patients on the basis of daily alcohol intake (n=90).

Grade of alcohol intake	<40 gm/d & / or <10 year	40-80 gm/d	81-160 gm/d	>160 gm/d	Non alcoholic	Total
Number	18	63	72	0	127	270
%	6.6	23.3	26.6	0	43.5	100

Table 4: Distributions of patients on the basis of viral marker positivity (n=90).

Viral marker	Number	%
HbsAg +ve	30	33.3
Anti HCV Ab +ve	3	3.3
Both +ve	0	0
Both –ve	57	63.3
Total	90	100

Biochemical: (Table 5 & 6) 80% have hyperbilirubinemia and 70% have conjugated hyperbilirubinemia. SGOT & ALP levels are increased in 56.6% cases but SGPT levels are increased in only 26.65% cases. In 30% cases albumin levels are less than 3 gm%. 53.3% cases have altered albumin- globulin ratio - may be due to chronic liver disease. Altered liver function in the form of increased prothrombin time - in 30% cases, which may increase risk of bleeding. 76.7% (40% ± 36.7%) of HCC patients have AFP level more than 500unit and practically diagnostic of HCC. But 23.3% have AFP level less than 500 units. All HCC patients have AFP level above the normal range of 10 ng/ml. 20% have increased leukocyte count - may be due to secondary infection or reactionary. 26.6% have decreased hemoglobin and in 40% of them have increased ESR. Azotemia - probably due to other causes unrelated to HCC. Electrolyte imbalance is not common in this study group.

Table 5: Distributions of biochemical abnormality in all patients (n=90).

Liver function test	Number	%
Normal bilirubin	18	20
Bilirubin ≤3 mg%	27	30
Bilirubin >3 mg%	45	50
Conjugated hyperbilirubinemia	63	70
Normal SGPT	66	73.3
Normal SGOT	39	43.3
Normal ALP	39	43.3
Increased SGPT	24	26.6
Increased SGOT	51	56.6
Increased ALP	51	56.6
Albumin <3 gm%	27	30
A:G <1	48	53.3
Prothrombin time	27	30
Total	525	583

Table 6: Distributions of other parameter after investigations in all patients (n=90).

Blood report	Number	%(n=30)
Leukocytosis	18	20
Hemoglobin <10 mg%	24	26.6
Increased ESR	36	40
Diabetes	6	6.6
Azotemia	3	3.3
Hypo/hypernatremia	0	0
Hypo/hyper kalemia	3	3.3
Total	270	100

Radiological: 66.6% have chronic liver disease, portal hypertension and splenomegaly-cirrhosis is a definite possibility. Most SOLs are single (53.3%) and mostly hyper echoic (83.3%). Either the SOLs are large and single or small and multiple-affecting mostly right lobe (53.3%). Abdominal lymphadenopathy is rare. Lung or brain parenchymal metastasis is rare - only3.3%. Pleural effusion is also not common and left sided pleural effusion is not present in this study group (Table 7).

Table 7: Distributions of radiological abnormalities in all patients (n=90).

Parameter	Number	%
Chronic liver disease	20	66.6
Portal hypertension	20	66.6
Splenomegaly	20	66.6
SOL 1 in number	16	53.3
SOL 2/3 in number	1	3.3
SOL >3 in number	13	43.3
Hypo echoic SOL	5	16.6
Hyper echoic SOL	25	83.3
SOL<3cm in diameter	13	43.3
SOL 3-5 cm	4	13.3
SOL >5cm	13	43.3
Mostly Rt lobe	16	53.3
Mostly Lt lobe	7	23.3
Rt & Lt lobe equal	7	23.3
Abdominal lymph node	1	3.3
Total	181	603

Endoscopy: 43.4% of HCC patients have esophageal varices, 3.3% have gastric erosion and 33.3% have normal esophageal findings. Although only 3.3% manifested hematemasis and 16.6% have melaena.

DISCUSSION

This is the first study from eastern India revealing detailed analysis of epidemiological clinical biochemical etiological and radiological features.

The pattern of HCC incidence by age is sometimes dependent on the geographic pattern or on etiologic factors.[6] The male preponderance is somewhat more in

our study (Male:Female - 9:1). The incidence of HCC peaks during fifth and sixth decade as in other studies. The population-based data shows a male to female ratio of 3:1-2:1.1.[7]

However, high preponderance of HCC in males reported in hospital-based data could suggest a gender bias in seeking medical treatment

The clinical presentation of HCC patients in our study was similar to other studies. The paraneoplastic syndrome was very rare in this series. Only one patient developed troublesome hypoglycemia. The rarity of paraneoplastic syndrome has been seen in other Indian series as well.[8-10] The symptom duration at diagnosis was mainly 1-3 months like other study from India.[9,10]

Chronic viral hepatitis due to either HBV or HCV is the main cause of HCC in most area worldwide.[11,12] One study from India showed that HBV is the predominant factor for the development of HCC, often related to mutant form of HBV.[13]

Our study showed similar etiological profile with Hepatitis B being the most common cause, followed by alcohol ingestion (cirrhotic range), unknown cause and Hepatitis C only in 3.3 %. 50% of patients ingested alcohol for >10 years and in amount which can cause chronic liver disease. 16.6% of patients have history of other risk factor like diabetes and hypertension, indicating nonalcoholic fatty liver disease, which is probably a risk factor for HCC. 33.3% 0f HCC patients are sero-positive for HBV and 3.3% are sero-positive for HCV. Only 4 patients presented with HCC without any alarming features (*de novo*).

No patient is positive for both and 63.3% of HCC patients are sero-negative for both. Therefore in our study alcoholic chronic liver disease is the main cause of HCC, next come chronic HBV infection, nonalcoholic fatty liver disease and chronic HCV infection.

In HCC the sensitivity of serum AFP ranges from 39% to 64%, specificity 76–91% and positive predictive value 9-32%.[14,15]

In another study from India, the prevalence of detectable AFP in patients with HCC was 51%.[16] however in our study 76.7% of HCC patients have AFP level more than 500 unit and practically diagnostic of HCC, suggesting a good role of AFP as diagnostic purpose.

CONCLUSION

The characteristics of HCC in eastern India are somewhat different from the rest of worlds. Alcohol and HBV infection are the two most important etiology prevailing here. Lifestyle modification is very much needed to curtail the intake of alcohol to reduce the burden of HCC in this part of world. On the other hand Mandatory vaccination against Hepatitis B will very much reduce the incidence of HCC.

ACKNOWLEDGEMENTS

We acknowledge the contribution from all patients.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. World Health Organization. World cancer report, 2014. In: WHO, eds. WHO Report. Geneva: WHO; 2014: Chapter 1.1.
2. Bruix J, Sherman M. Management of hepatocellular carcinoma. Hepatology. 2005;42:1208-36.
3. ICMR. Annual report. In: ICMR, eds. National Cancer Registry Programme. New Delhi: Indian Council of Medical Research; 2008.
4. Jayant K, Rao RS, Nene BM, Dale PS. Rural cancer registry at Barshi - report 1988-92. In: Jayant K, Rao RS, Nene BM, Dale PS eds. A Report. Barshi: Rural Cancer Registry; 1994.
5. Bruix J, Sherman M, Llovet JM, Beaugrand M, Lencioni R, Burroughs AK, et al. Clinical management of hepatocellular carcinoma. Conclusions of the Barcelona-2000 EASL Conference. J Hepatol. 2001;35:421-30.
6. Bosch FX. Global epidemiology of hepatocellular carcinoma. In: Okuda K, Tabor E, eds. Liver Cancer. 1st ed. New York: Churchill Livingstone; 1997: 13-27.
7. Dinshaw KA, Rao DN, Shroff PD. Hospital cancer registry: annual report 1994. In: Dinshaw KA, Rao DN, Shroff PD, eds. A Report. Mumbai: Tata Memorial Hospital; 1997.
8. Sarin SK, Thakur V, Guptan RK, Saigal S, Malhotra V, Thyagarajan SP, et al. Profile of hepatocellular carcinoma in India: an insight into the possible etiologic associations. J Gastroenterol Hepatol. 2001;16:666-73.
9. Singh SV, Goyal SK, Chowdhury BL. Primary carcinoma of liver in Udaipur. J Assoc Physicians India. 1971;19:693-5.
10. Agarwal AK, Manvi KN, Mehta JM, Shah MJ. Clinical diagnosis of hepatoma. J Assoc Physicians India. 1966;14:465-8.
11. Okuda M. Hepatocellular carcinoma: recent progress. Hepatology. 1992;15:948-63.
12. Verner S. Viruses and hepatocellular carcinoma. Gut. 1994;35:825-32. Omata M, Ashcavai M, Liew C-T, Peters RL. Hepatocellular carcinoma in the USA, etiologic consideration. Localization of hepatitis B Ags. Gastroenterology. 1979;76:279-87.
13. Sarin SK. Diagnostic paradox of hepatocellular carcinoma. J Natl Cancer Inst. 1995;22:23.
14. Collier J, Sherman M. Screening for hepatocellular carcinoma. Hepatology. 1998;27:273-8.

15. Sherman M. Alpha-fetoprotein: an obituary. J Hepatol. 2001;34:603-5.
16. Hill PG, Johnson S, Madangopalan N. Serum alpha-fetoprotein and hepatitis B antigen in subjects with hepatoma in south India. Indian J Med Res. 1977;65:482-7.

Cite this article as: Mukherjee S, Dhar K, Datta S, Mukherjee AK. Hepatocellular carcinoma in eastern India, a detail analytical report from a tertiary care hospital. Int J Sci Rep 2015;1(1):69-73.