

# Hepatocellular Carcinoma presenting as a abdominal pain -A Case Report

Arun Kumar<sup>1</sup>, Bibek Poudel<sup>2</sup>,

<sup>1</sup> Associate Professor, Department of Biochemistry, Manipal College of Medical Sciences, Pokhara, Nepal

<sup>2</sup> Lecturer, Department of Biochemistry, Manipal College of Medical Sciences, Pokhara, Nepal

**Correspondence:**

✉ [arun732003@gmail.com](mailto:arun732003@gmail.com)  
[bibekclb@yahoo.com](mailto:bibekclb@yahoo.com)

**1** Mobile Number:  
+977-9816664537

**2** Mobile Number:  
+977-9841553607

## Abstract

Cancer is the leading cause of death in economically developed countries and the second leading cause of death in developing countries. It is observed that 70% of cancer deaths occur in low- and middle income countries. Of these cancers, 30% of can be prevented.

The etiological factors associated with hepatocellular carcinoma vary from country to country. It most commonly affects adults, children who are affected with biliary atresia, infantile cholestasis, glycogen-storage diseases and other cirrhotic diseases of the liver are predisposed to developing hepatocellular carcinoma.

The current case report discusses about the diagnosis of hepatocellular carcinoma through elevated levels of alpha-feto protein (AFP) though presented with abdominal pain and was confirmed with supportive findings of CT scan and ultrasound.

**Key words:** Hepatocellular carcinoma, Alpha-feto protein, abdominal pain, NLR ratio.



This article is available from:  
[www.jbiomed.com](http://www.jbiomed.com)

## Introduction

Cancer is the leading cause of death in economically developed countries and the second leading cause of death in developing countries [1]. In 2008, around 12.7 million cases were detected and 7.6 million people died from cancer worldwide [2]. Of these, 56% of the cases and 64% of the deaths occurred in the economically developing world. The burden of hepatocellular cancer is increasing in economically developing countries not only due to rapid population aging and growth, but also the adoption of cancer-associated lifestyle choices including smoking and physical inactivity [3]. It is observed that 70% of cancer deaths occur in low- and middle income countries [4]. Of these cancers, 30% of can be prevented.

Though it varies widely from country to country, the major risk factors for hepatocellular carcinoma (HCC) are alcoholism, Hepatitis B and C, cirrhosis of liver, hemochromatosis,

Wilson's disease and type-2 diabetes. In countries like China, Hepatitis B would be the predominant cause of HCC. The risk of HCC in type-2 diabetes is greater (from 2.5 to 7.1 times to non-diabetic risk) [5]. Although HCC most commonly affects adults, children who are affected with biliary atresia, infantile cholestasis, glycogen-storage diseases and other cirrhotic diseases of the liver are predisposed to developing hepatocellular carcinoma.

## Case Report

A sixty-five year old male from Magar Community of Dhaulagiri zone and western region of Nepal visited Manipal Teaching Hospital with the chief complaint right-sided cramping abdominal pain. History and examination revealed that the patient was a long-term smoker, having 15-20 cigarettes per day for the past 20 years. He was also an alcoholic with moderate consumption of 400-600ml of home-made liquor per

day. There was no family history of cancer or any other liver disorders in the family. The patient was on adequate nutrition as he did not show any sign of nutritional deficiency. He was not well educated but literate. On further questioning it was revealed that the consumption of alcohol and inclination towards the non-vegetarian diet is a part of the practice of the patient's local community. Anthropometric examination was unremarkable and the patient had no recent history of nausea and vomiting. Heart rate, respiratory rate and neurological examination were normal, and the patient was not hypertensive. Subsequently, the following biochemical tests were performed:

The biochemical parameters namely FBS: 93 mg/dl (<100), Urea: 31mg/dl (15-40), Creatinine: 1.3 mg/dl (<1.5), Na<sup>+</sup>: 138mEq/L (135-150), K<sup>+</sup>: 3.9 mEq/L (3.5-5), Ca<sup>++</sup>: 8.5 mg/dl (8.5-10.4), PO<sub>4</sub><sup>---</sup>: 3.9 mg/dl (2.7-4.5), Total Bilirubin: 0.8 mg/dl (<1), Direct Bilirubin: 0.3 mg/dl(<0.3), Indirect Bilirubin: 0.5 mg/dl (<0.7), AST: 63 U/L (<40), ALT: 58U/L (<40), Alp: 66 U/L (<195), γ-GT: 63 U/L (<50), Total Protein: 6.3 g/dl (6-8), Albumin: 2.7g/dl (3.2-5.5), Globulin: 3.6 mg/dl (2.5-3), AG Ratio: 0.75 (1.2-2:1), Total Cholesterol: 195 mg/dl (<200), HDL-C: 42 mg/dl (30-60), LDL-C: 118 mg/dl (70-130), VLDL: 35mg/dl (10-50), TG: 165 mg/dl (40-1700, Uric Acid: 4.5 mg/dl (3.5-7.2).

Hematological parameters namely Hb: 15 mg/dl (>13), WBC: 8300 (4000-11000), N=70, L=25, M=4, E=1, NLR: 2.8, ESR: 21mm/hr (<10).

Serological findings including hepatitis B and C were found to be non-reactive.

Urine and stool routine and microscopic examination were normal.

Ultrasound finding revealed normal anatomy of the gall bladder, spleen, pancreas, kidney, urinary bladder and prostate with no lymphadenopathy noted in the abdominal cavity. However, ultrasound of the liver revealed heterogeneously hyperechoic lesions in the right lobe of liver (11X9.6 cm) and one hypoechoic lesion (2.5 cm) within the left lobe of liver. The overall impression of this ultrasound report was multiple lesions seen within liver, suggestive of metastasis.

For further confirmation, the patient was advised to go for NECT and CECT scan of abdomen and pelvis. The findings included: enlarged liver (18.9 cm). Lobulated outline hypodense lesion seen involving right lobe of liver. No evidence of calcification within. Post contrast scan shows heterogeneous enhancement of the mass lesion with no-enhancing hypodense areas suggestive of necrosis. It measures approximately 12.8X9.4 cm in size. Inferomedially, fat plane with adrenal

gland and upper pole of kidney is lost. Hypodense filling defect suggestive of thrombus is seen within intrahepatic and extrahepatic inferior vena cava above renal hilum. Portal and hepatic veins are normal. No dilatation of IHBR.

Gallbladder is not visualized. CBD appears normal.

Pancreas, spleen, bilateral kidney, adrenal glands, abdominal viscera, major abdominal vessels, urinary bladder, prostate glands, visualized soft tissues and visualized section of chest appears normal.

The case was suspected of hepatocellular carcinoma as the liver was enlarged with a heterogeneously enhancing mass lesion within the right lobe of the liver. With ultrasound and CT scan findings being suggestive of hepatocellular carcinoma, a further blood sample was sent to the biochemistry lab for α-feto protein (AFP) measurement. The result reveals four-fold elevation of α-feto protein (AFP) level showing a value of 32 U/L (0.0-8.5).

## Discussions

In this case study, it was observed that the levels of total protein decreased with concomitant decrease in albumin levels and increased in globulin levels. Also the aspartate aminotransferase, alanine aminotransferase and gamma-glutamyltransferase levels were drastically increased. The findings were comparable with a prospective cohort of 1822 patients with anti-HCV seronegative chronic HBV carriers where, they reported [6] AST, ALT, AFP, GGT, total bilirubin, total protein, albumin, globulins, apolipoprotein A-1 and apolipoprotein B were increased. The case also observed high neutrophil to lymphocyte (N/L) ratio which was 2.8. Usually the NLR is a simple marker of subclinical inflammation and has been used to predict the outcomes in patients with cancer and coronary artery disease [7]. This ratio integrates the information on two different immune pathways- the neutrophils that are responsible for ongoing inflammation and the lymphocytes that represent the regulatory pathway, thus indication of the overall inflammatory status of the body. A lower ratio is predictive of good outcomes compared to a higher ratio. Previous studies have shown that low N/L ratio is associated with smoking and higher body mass index (BMI).

Alpha-feto protein is a glycoprotein with molecular weight of 70 kd with 591 amino acids. The gene coding for AFP is located in the q arm of chr 4 (4q25). It is synthesized by fetus hepatocytes, yolk sac and also in the gastrointestinal tract and is structurally analogous to albumin of adults. During the later time of life AFP is completely replaced by albumin and principally absent in blood. The levels of AFP decline rapidly

after birth but invariably the reduction begins at the end of first trimester. However, it is produced by adult hepatocytes when affected by hepatocellular carcinoma.

AFP is found to be elevated in various clinical disorders, including primary hepatocellular carcinoma, germ cell tumors and metastatic cancers.

In this case study we found the levels of AFP was 32 U/L which was four-fold higher the upper reference limit. Hence, in conjunction with the ultrasound report and CT scan, the presence of tumour marker (AFP) strongly supports the findings of primary hepatocellular carcinoma.

Tai et al [8] reported that AFP is important in diagnosis of HCC but it may be also increased in case of hepatitis B and hepatitis C viral infection. In the current case report we found hepatitis B and C virus non-reactive. This further supports the conclusion that elevated AFP levels was the result of HCC rather than an alternative diagnosis.

Since AFP is positively correlated with CT scan and Ultrasound findings we recommend the level of AFP as a hepatic biomarker to establish and support the diagnosis of HCC.

## Acknowledgement

Jennifer Patten  
MBBS Final Year  
University of East Anglia  
United Kingdom  
[Jennifer.patten@uea.ac.uk](mailto:Jennifer.patten@uea.ac.uk)

Follow us:



**Medicalia.org**

Where Doctors exchange clinical experiences, review their cases and share clinical knowledge. You can also access lots of medical publications for free. Join Now!

<http://medicalia.ning.com/>

## References

1. American Joint Committee on Cancer. AJCC Cancer Staging Manual. 7<sup>th</sup> Ed. New York: Springer. 2010.
2. Jemal, A., Bray, F., Melissa, M., Ferlay, J., Ward, E., Forman, D. Global Cancer Statistics. CA Cancer J Clin 2011; 61: 69-90.
3. Thun, MJ., DeLancey, JO., Center, MM., Jemal, A., Ward, EM. The global burden of cancer: priorities for prevention. Carcinogenesis 2010; 31 (1): 100-110.
4. Tfayli, A., Temraz, S., Mrad, RA., Shamseddine, A. Breast Cancer in Low- and Middle-Income countries: An emerging and challenging epidemic. Journal of Oncology 2010.
5. Zachary, T. Bloomgarden. Aspects of Type 2 Diabetes and Related Insulin-Resistant States. Diabetes Care 2006; 29 (3): 732-740.
6. Lin, YJ., Lee, MH., Yang, HI., Jec, CL., You, SL., Wang, LY., Lu, SN., Liu, J., Chen, CJ. Predictability of liver-related seromarkers for the risk of hepatocellular carcinoma in chronic hepatitis B patients. PLoS One 2013; 8 (4).
7. Chen, TM., Lin, CC., Huang, PT., Wen, CF. Neutrophil-to lymphocyte ratio associate with mortality in early hepatocellular carcinoma patient after radiofrequency ablation. J Gastroenterol hepatol 2012; 2793: 553-561.
8. Tai, WC., Hu, TH., Wang, JH., Hung, CH., Lu, SN., Changchien, CS., Lee, CM. Clinical Implications of alpha-fetoprotein in chronic hepatitis. C. J Formos Med Assoc 2009; 108 (3): 210-218.

Publish with iMedPub

<http://www.imedpub.com>

- ✓ JBS publishes peer reviewed articles of contemporary research in the broad field of biomedical sciences. Scope of this journal includes: Biochemistry, Biomedical sciences, Biotechnology, Microbiology, Molecular biology and Genetics. Secondary research including narrative reviews, systematic reviews, evidencebased articles, meta-analysis, practice guidelines will also be considered for publication.
- ✓ From time to time invited articles, editorials and review of selected topics will be published.
- ✓ The editorial board of JBS shall strive to maintain highest standards of quality and ethics in its publication.

Submit your manuscript here:  
<http://www.jbiomeds.com>