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Case Report

Deadly Phase of HCV Related CLD with Hepatocellular Carcinoma

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ABSTRACT

Introduction-.HCV-related hepatocellular carcinoma (HCC) is a major cause of cancer mortality, often arising 20–40 years after infection, almost always in the presence of cirrhosis. While direct-acting antivirals (DAAs) offer a high cure rate (sustained virological response, SVR), reducing HCC risk, it is not completely eliminated. HCV induces chronic inflammation, oxidative stress, and cirrhosis, which drive carcinogenesis. The risk factors are increased risks include male gender, older age at infection, alcohol abuse, metabolic syndrome (diabetes, steatosis), and genotype 1b or 3. HCV related HCC usually presents with pain abdomen in advanced stage but in early stage it can remain asymptomatic. The pain is usually dull aching, in right hypochondrium and may radiate to back. It can be associated with anorexia, weight loss or jaundice. Patients with HCV-associated cirrhosis require regular monitoring with six monthly alpha-feto protein level (AFP) and abdominal ultrasound.

Case Report- We report a sixty-six-year-old male, not a known case of any chronic illness, non-smoker, non-alcoholic presented with short history of vague abdominal pain, mainly in epigastric area for last two months and abdominal distension for last one month. On biochemical evaluation complete hemogram revealed pancytopenia, liver function test was deranged i.e. there was mild hyper bilirubinaemia, transaminitis, hypoproteinaemia and hypoalbunemia. The complete lipid profile was in below normal range but renal function test, serum electrolytes and blood sugar level were in normal range. The ultrasonogram abdomen showed altered echotexture with moderate ascites and suspicious lesions in both lobes of liver. The upper gastro-intestinal endoscopy revealed grade one oesophageal varices. The AFP level was significantly raised to 2148 IU/ml. Hence, to rule out hepatocellular carcinoma (HCC), triple phase computed tomography scan which showed irregular liver border with relative hypertrophy of caudate lobe, splenomegaly with multiple collaterals, suggestive of portal hypertension. There were multiple lesions in both lobes of liver which showed hyperenhancement on arterial phase and washout in venous phase. The largest lesion was 91 x 86 mm with extension into right branch of portal vein- likely of HCC. There was mild pericardial effusion, cholelithiasis with multiple abdominal retroperitoneal lymphadenopathies. His viral screen showed anti HCV antibody positivity with HbsAg and anti-HIV antibody negativity. His HCV RNA quantitative showed high viral load- 2 x 10^9 IU/ml. He was started on 24-week antiviral treatment with sofosbuvir 400 mg and Velpatasvir 100 mg, along with diuretics and other supportive therapy. The surgical opinion was taken who advised for conservative management and supportive therapy only, as he was out of liver transplantation, surgical resection and trans arterial chemoembolization (TACE).

Conclusion- HCV patients can have uncommon or atypical presentations with very short history. Every HCC patient has to be mandatory evaluated for Hepatitis B, C and HIV, along with other aetiological factors. Pain abdomen can be first manifestation of HCC in limited number of patients, as in our case which was due to portal vein invasion by HCC. He had very short downward hill course of two

months only, with overall predicted survival rate of six months only. Hence, it is wisely said that prevention is better than cure and same applies to HCV and HBV infections.

Keywords*: HCV, Chronic liver disease, Hepatocellular carcinoma, Portal vein, Triple phase CT scan, Alpha fetoprotein level.*

INTRODUCTION

Chronic hepatitis C virus (HCV) infection is a major cause of hepatic fibrosis and cirrhosis, with a risk for the development of hepatocellular carcinoma (HCC). Although highly effective direct-acting antivirals (DAAs) are available, the incidence, morbidity, and mortality of HCV-associated HCC are still high **[1].** HCV is an uncommon for RNA viruses, as it manages to persist in up to 80% of cases, leading to chronic hepatitis C (CHC) with a high risk of developing fibrosis, cirrhosis, and eventually hepatocellular carcinoma (HCC). Annually, 1–4% of cirrhotic CHC patients develop HCC, the most common form of primary liver cancer, which has the third highest mortality among all cancer entities **[2,3]**. While cirrhotic liver disease generally increases the risk of developing neoplastic lesions, the annual incidence of de novo HCC is reported to be up to twofold higher in patients with HCV-related cirrhosis compared to other etiologies, such as autoimmune hepatitis **[4]**, metabolic-associated fatty liver disease, and alcoholic fatty liver disease **[5]**. Furthermore, HCV-associated HCC can develop even in the absence of cirrhosis **[6]**. Thus, HCV poses a specific and significant risk for cancer development.

CASE REPORT

We report a sixty-six-year-old male, not a known case of any chronic illness, non-smoker, non-alcoholic presented with short history of vague abdominal pain, mainly in epigastric area for last two months and anorexia & abdominal distension for last one month. On general physical examination, he was lean, thin, cachexic and anaemic. The abdominal examination revealed ascites and rest of systemic examination was essentially normal. On biochemical evaluation complete hemogram revealed pancytopenia, liver function test was deranged i.e. there was mild hyper bilirubinaemia, transaminitis, hypoproteinaemia and hypoalbunemia. The complete lipid profile was in below normal range but renal function test, serum electrolytes and blood sugar level were in normal range. The ultrasonogram abdomen showed altered echotexture with moderate ascites and suspicious lesions in both lobes of liver. The upper gastro-intestinal endoscopy revealed grade one oesophageal varices. The AFP level was significantly raised to 2148 IU/ml. Hence, to rule out hepatocellular carcinoma (HCC), triple phase computed tomography scan which showed irregular liver border with relative hypertrophy of caudate lobe, splenomegaly with multiple collaterals, suggestive of portal hypertension. There were multiple lesions in both lobes of liver which showed hyperenhancement on arterial phase and washout in venous phase. The largest lesion was 91 x 86 mm with extension into right branch of portal vein- likely of HCC. There was mild pericardial effusion, cholelithiasis with multiple abdominal retroperitoneal lymphadenopathies. His viral screen showed anti HCV antibody positivity with HbsAg and anti-HIV antibody negativity. His HCV RNA quantitative showed high viral load- 2 x 10^9 IU/ml. He was started on 24-week antiviral treatment with sofosbuvir 400 mg and Velpatasvir 100 mg, along with diuretics and other supportive therapy. The surgical opinion was taken who advised for conservative management and supportive therapy only, as he was out of liver transplantation, surgical resection and trans arterial chemoembolization (TACE).

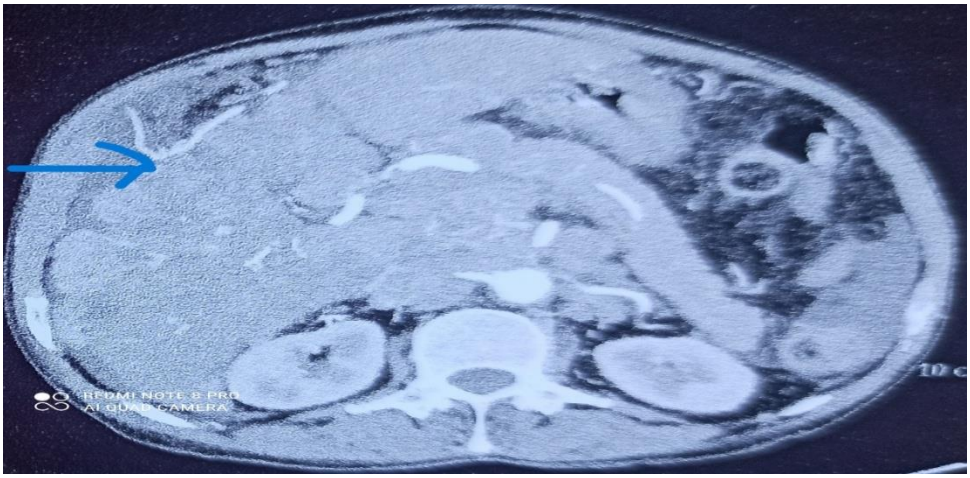

Figure 1- Triple Phase CT Scan abdomen showing enhancing lesions in both lobes of Liver (blue arrow) in arterial phase suggestive of HCC in HCV related CLD Patient.

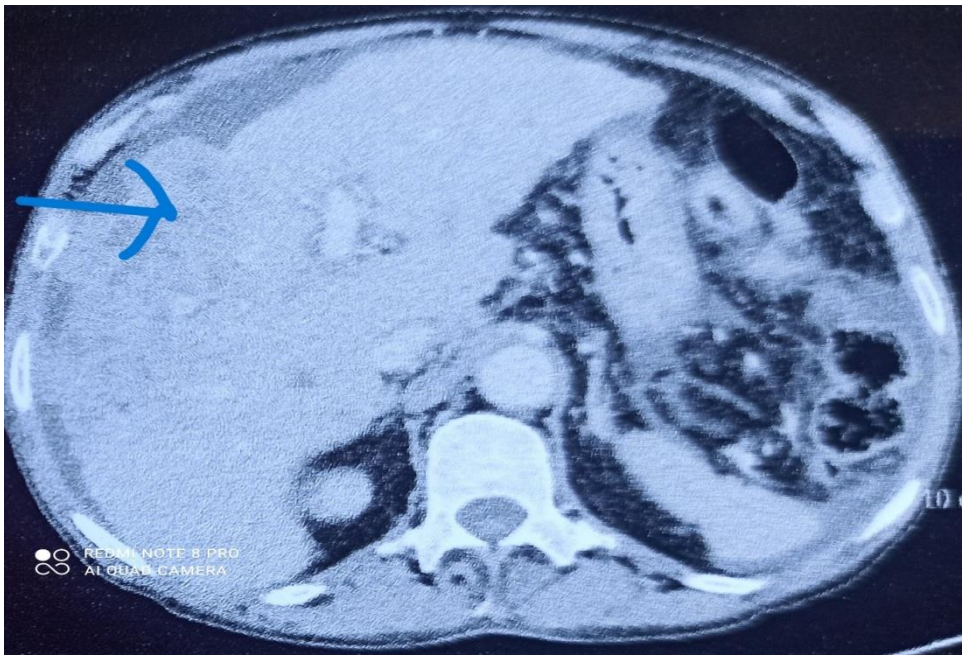

Figure 2- Triple Phase CT Scan abdomen showing complete washout of lesions in both lobes of liver (blue arrow) in venous phase suggestive of HCC in HCV related CLD Patient.

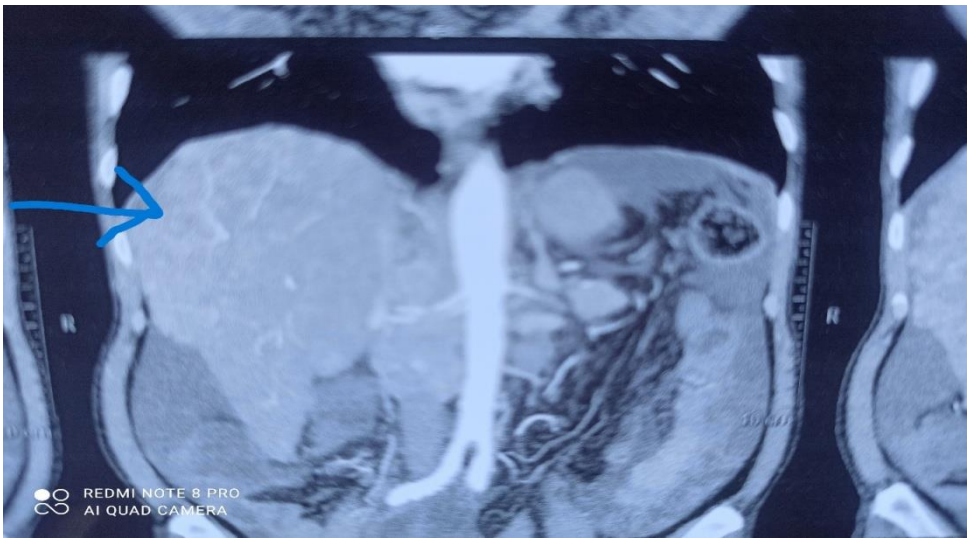

Figure 3- Triple Phase CT Scan abdomen showing mild washout of lesions in both lobes of liver (blue arrow) in portal phase suggestive of HCC in HCV related CLD Patient.

DISCUSSION

Hepatitis C virus (HCV) is an RNA virus responsible for liver inflammation and the development of hepatocellular carcinoma (HCC). It is estimated that 1.5 million individuals are infected with HCV every year. However, only 20%–30% develop liver cirrhosis, of which 1%–4% develop HCC. Cancer induction appears to be rather indirect, crucially involving chronic inflammation and the establishment of a pro-tumorigenic environment, with a potentially stable and self-sustaining metabolic and epigenetic reprogramming. The remaining risk for patients to develop HCC even after SVR, hence, appears to be due to a sustainable impact on liver homeostasis as a consequence of infection. While direct interactions of HCV RNA or proteins with oncogenic pathways within the infected hepatocytes themselves might still contribute to carcinogenesis to a certain degree **[7, 8]**. HCV-associated HCC typically develops after years to decades of chronic infection. Ongoing elimination of infected hepatocytes by immune cells and cell death upon virus-induced stress continuously trigger regenerative hepatocyte proliferation **[9]**. The inherent risk of genetic mutations due to erroneous DNA replication is aggravated by oxidative damage **[10]**. This is mainly caused by reactive oxygen species (ROS), secreted by immune cells **[11]** or released from the mitochondria of infected hepatocytes **[11,12)** There is evidence that in addition to chronic inflammation, HCV directly contributes to fibrogenesis. For patients treated in advanced stages of liver disease, the American Association for the Study of Liver Diseases (AASLD) recommends surveillance for HCC in SVR patients with cirrhosis **[13]**, while the European Association for the Study of the Liver (EASL) recommends

screening already from advanced fibrosis (METAVIR score F3) onwards **[14]**. Hepatocellular carcinoma (HCC) is more common in the right lobe of the liver. HCC is most common in segment 8 (the superior-posterior segment of the right lobe). The right lobe is more frequently affected due to its larger size and the higher volume of blood it receives from the superior mesenteric vein, which carries pathogens and toxic substances directly into the right liver parenchyma, potentially causing higher rates of cirrhosis, which is a major precursor to HCC. Though rare, HCC can occur in an accessory liver lobe, with most reported cases also located on the right. HCV related HCC usually presents with pain abdomen in advanced stage but in early stage it can remain asymptomatic. The pain is usually dull aching, in right hypochondrium and may radiate to back. It can be associated with anorexia, weight loss or jaundice. The pain abdomen can have various reasons like liver capsule stretching, portal vein invasion by tumour or tumour rupture. In our case also main symptom was pain abdomen was dull aching, as it was in advanced stage and was additionally having invasion in right branch of portal vein. It was also associated with anorexia, weight loss and later followed by formation of ascites which presented as abdominal distension.

CONCLUSION

HCV patients can have uncommon or atypical presentations with very short history. Every HCC patient has to be mandatory evaluated for Hepatitis B, C and HIV, along with other aetiological factors. Pain abdomen can be first manifestation of HCC in limited number of patients, as in our case which was due to portal vein invasion by HCC. He had very short downward hill course of two months only, with overall predicted survival rate of six months only. Hence, it is wisely said that prevention is better than cure and same applies to HCV and HBV infections.

CONFLICT OF INTEREST

The authors declare that there was no conflict of interest or any kind of funding was taken for publishing this case report.

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