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Case report Open Access

HBV Related HCC Presenting as Rupture- An Uncommon Presentation

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1. Abstract

1.1. Introduction

Hepatitis B virus (HBV) impacts large number of populations worldwide and has both hepatic and extrahepatic manifestations. It can present as acute hepatitis, chronic hepatitis, cirrhosis and hepatocellular carcinoma (HCC). HBV infection is associated with the risk of developing HCC with or without an underlying liver cirrhosis, due to various direct and indirect mechanisms promoting hepatocarcinogenesis. Hepatocellular carcinoma (HCC) rupture related to hepatitis B virus (HBV) infection can present with severe abdominal pain, hypotension, and abdominal distension, often accompanied by hemoperitoneum

1.2. Case Report

We report a fifty-five-year-old male, a known case of chronic hepatitis B related chronic liver disease on regular antiviral treatment for last five years. He presented with acute pain abdomen for last four days which was being symptomatically treated with injectable analgesics and proton pump inhibitor but for no relief. His ultrasonogram showed some benign lesion in segment six of liver. At this stage, he reported back to our department for follow up. His ultrasonogram was repeated which showed a mass lesion in liver with massively raised Alpha feto protein of 500 I.U. His triple phase computed tomography scan showed a tumour lesion with rupture and collection in para colic gutter.

1.3. Conclusion

Hepatitis B has many presentations varying from inactive carrier stage in majority to cirrhosis and H.C.C but HCC presenting for first time with tumour rupture is not common but should be kept in mind in cirrhotic patients presenting with acute pain abdomen.

2. Introduction

HBV infection has become major health problem in developing country like India which has many hotspots like Haryana, Punjab, Uttar Pradesh, Uttarakhand, North eastern states and Hepatitis B Surface Antigen (HbsAg) positivity varies between 2–4.7% [1,2]. In India, approximately 40 million people are chronically infected with Hepatitis B [3]. The major routes of transmission of Hepatitis B include vertical transmission, unsafe needle & sexual practices, repeated exposure to blood & blood products like who receive repeated transfusion of blood, are on maintenance haemodialysis, intravenous drug abusers, males having sex with male, female sex workers, sexual partners & care takers of HBV patient and prisoners [4]. The molecular profile of HBV-HCC is extensively and continuously under study, and it is the result of altered molecular pathways, which modify the microenvironment and lead to DNA damage. HBV produces the protein HBx, which has a central role in the oncogenetic process. Proper management of the underlying HBV-related liver disease is fundamental, including HCC surveillance, viral suppression, and application of adequate predictive models. When HBV-HCC occurs, liver function and HCC characteristics guide the physician among treatment strategies [5].

3. Case Report

We report a fifty-five-year-old male, a known case of chronic hepatitis B related chronic liver disease on regular antiviral treatment for last five years. He presented with acute pain abdomen for last four days which was being symptomatically treated with injectable analgesics and proton pump inhibitor but for no relief. At this stage, he reported back to our department for follow up. There was no history of fever, weight loss, haematemesis, melena, altered sleep pattern or behaviour, bladder or bowel symptoms, breathlessness on exertion or rest. On biochemical evaluation he had pancytopenia on complete hemogram, liver function test was deranged i.e. there was low albumin level, mild hyperbilirubinemia, raised transaminitis with reversal of ratio of AST being more than ALT and increased INR. The lipid profile showed lower values of all parameters including total cholesterol, triglycerides, LDL, VLDL and HDL levels. His renal function test, serum electrolytes and blood sugar level were in normal range. The viral screen was positive for HbsAg and anti HCV antibody, anti-HIV antibody,

Serum IgM HAV & HEV antibody test were negative, Alpha feto protein level (AFP) was significantly raised to 500 I.U.and HBV DNA quantitative was Eighty thousand I.U/ml. His ultrasonogram showed altered echotexture of liver with some benign lesion in segment six of liver and splenomegaly. There was no ascites but portal vein diameter was more i.e. 12mm. His ultrasonogram was repeated which showed altered echotexture of liver with a mass lesion in segment six. His triple phase computed tomography scan showed a heterogenous lesion with arterial enhancement and venous wash off with subcapsular rupture and collection in para colic He was married and on family screening, all her family members were found to be HbsAg negative, thus were vaccinated with complete course of HBV vaccination. Patient was referred to surgical oncologist by whom intra-abdominal drain was put but patient later on left against medical advice and was lost on follow up.

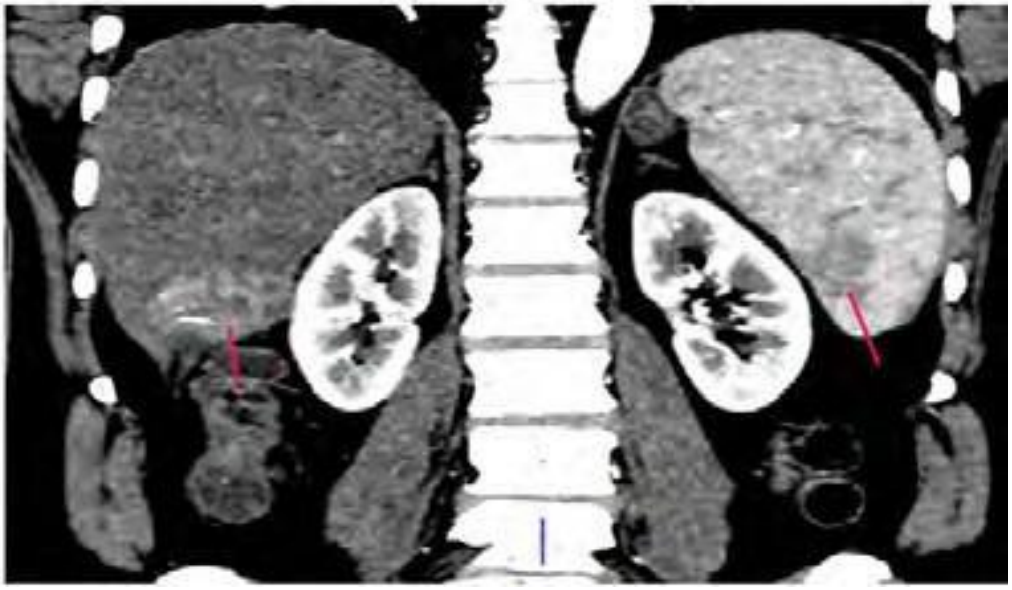

Figure 1: Showing Contrast enhanced CT arterial phase images in coronal and axial plane show a relatively encapsulated arterial phase heterogeneously enhancing lesion in segment VI of liver.

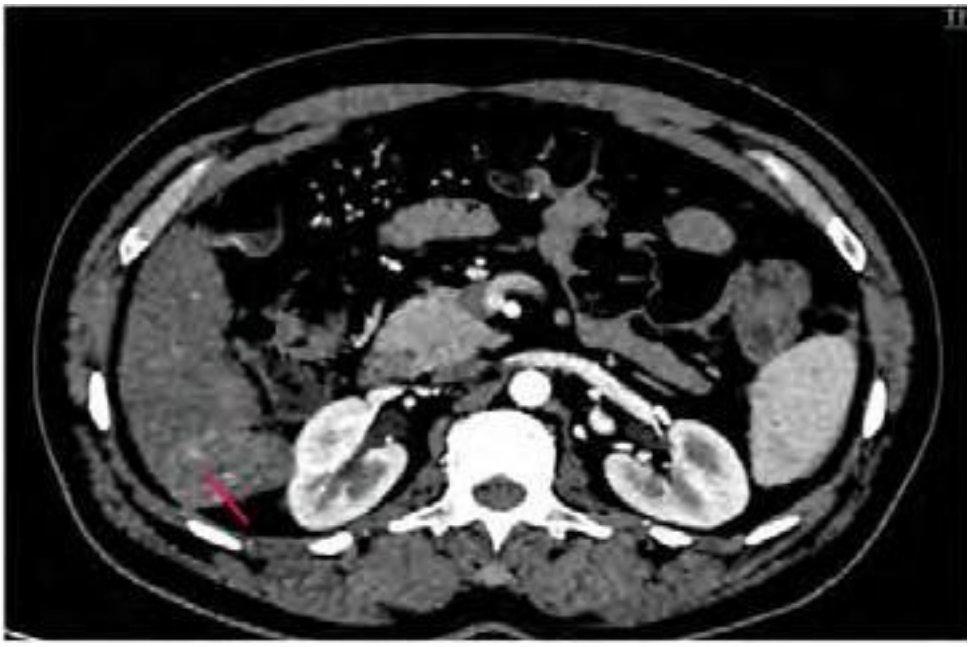

Figure 2: Showing Hepa cellular Carcinoma in Arterial Phase.

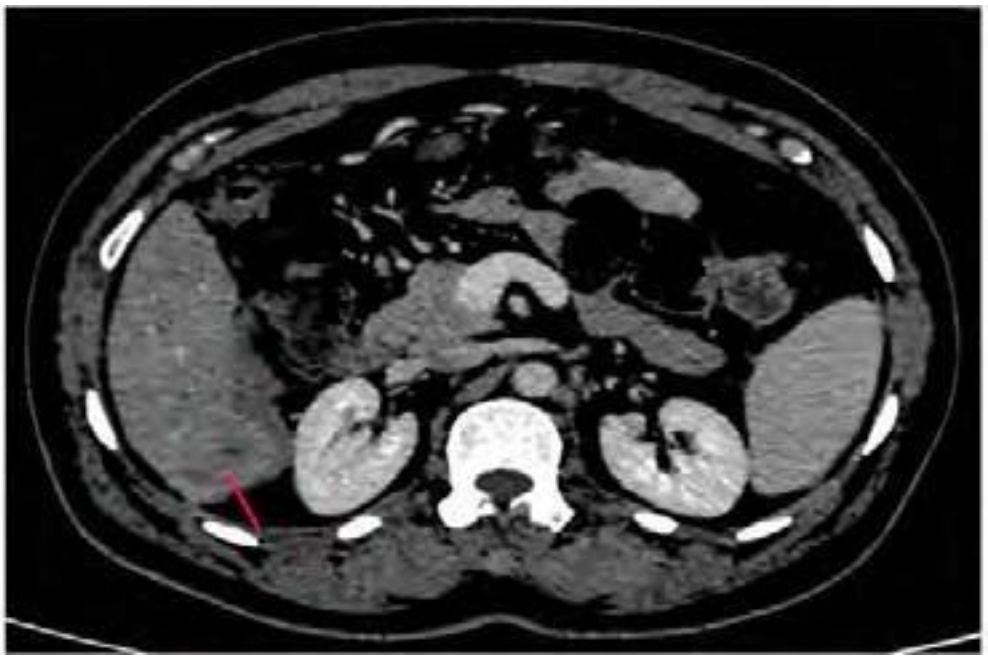

Figure 3: Showing Contrast enhanced CT portal venous phase axial images showing mild washout in the segment VI liver lesion with high density collection along the subcapsular aspect.

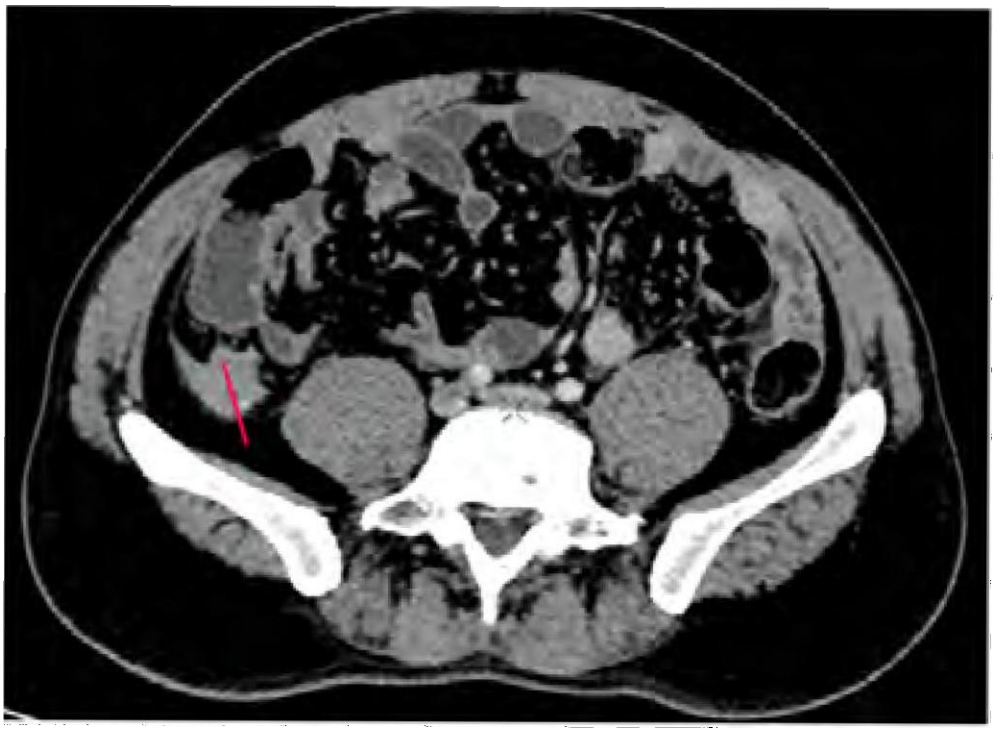

Figure 4: High Density Collection in Right Paracolic Gutter.

4. Discussion

The World Health Organization (WHO) aims at reducing HBV infections by 90% and increasing global vaccine coverage to 90% [6] for which health awareness is mandatory regarding hepatitis B prevention, screening, and vaccination [7]. The HBV infection behaves like tip of iceberg where 90% of patients are unaware about it, thus remain undiagnosed and in future can progress to cirrhosis, and HCC [8]. Liver cancer is important cause of mortality associated with cancer pan globally with annual death toll of 700,000 [9]. Hepatocellular Carcinoma (HCC) represent the major variety of primary liver malignancies and is responsible for 70% to 85% of the total liver cancer burden [10]. The maximum cases of hepatocellular carcinoma (75% to 90%) develop in cirrhotic liver caused by various factors like chronic HBV & HCV, alcohol, obesity and diabetes mellitus, autoimmune hepatitis or hemochromatosis [11-13]. In last three decades, about 63% increase in total deaths has been reported globally because of viral hepatitis HBV & HCV infections because it leads to continuous liver damage which gradually progresses to cirrhosis and H.C.C [14]. HCC prevention in the contest of CHB can be primary, secondary, and tertiary. Data on HBV treatments suggests that a significant amount of HCC cases could be avoided through secondary prevention [15]. Secondary prevention of HCC consists of the treatment of underlying liver diseases aiming at the prevention of disease progression [16]. The available therapies for HBV are mainly divided into two typologies: IFNs (interferons), and

NUCs (nucleoside/nucleotide analogs). Hepatocellular carcinoma (HCC) rupture related to hepatitis B virus (HBV) infection can present with severe abdominal pain, hypotension, and abdominal distension, often accompanied by hemoperitoneum. The rupture can lead to a life-threatening condition requiring prompt diagnosis and treatment. The clinical presentation includes abdominal pain which can be sudden, severe abdominal pain is the most common symptom, reported in 66-100% of cases. Haemorrhagic shock can occur in 33-90% of patients due to hemoperitoneum, that can lead to abdominal distension in about 33% of cases and can be detected through diagnostic paracentesis or imaging. Progressive anaemia can occur due to blood loss, leading to symptoms like paleness, dizziness, and fatigue. Jaundice and ascites may also be present due to the association of HCC with cirrhosis. In some cases, haemorrhage can be slow and progressive, leading to anaemia without obvious signs of peritoneal irritation. The diagnosis depends upon Contrast-enhanced CT or MRI scans which are crucial for confirming the rupture, visualizing active bleeding (contrast extravasation), and assessing the extent of the tumour and hemoperitoneum. Abdominal paracentesis can help detect hemoperitoneum and guide further management. First step of management is achieving haemostasis and treatment may involve emergency surgery, trans arterial embolization (TAE), or a combination of both. Emergency laparotomy may be necessary to control bleeding, particularly in unstable patients. Trans arterial Embolization (TAE) with or without chemoembolization, is a minimally invasive approach that can effectively control bleeding. In hemodynamically stable patients, conservative management with close monitoring and correction of coagulopathy may be an option. In some cases, staged hepatectomy (liver resection) may be considered after initial haemostasis and stabilization. Ruptured HCC is associated with high mortality, but active treatment can improve outcomes. The prognosis depends on factors like tumour size, liver function, and the extent of bleeding. While some studies suggest that rupture may not significantly increase metastasis risk, others indicate that peritoneal metastasis can occur after rupture. The incidence of HCC rupture is higher in Asia and Africa than in Europe. In Asia approximately 10% of patients with a diagnosis of HCC die due to rupture each year. Spontaneous rupture is the third most common cause of death due to HCC after tumour progression and liver failure. Usually, sudden development of severe pain abdomen in cirrhotic hints at portal vein thrombosis (PVT), which may or may not be associated with development of H.C.C. The rupture of HCC is reported but is not as commonly seen as PVT. The management is easier in PVT and outcome is also better than tumour rupture. In our case, uncommon thing was that in previous ultrasonogram done six months before, there was no evidence of HCC and patient in background of Cirrhosis with acute pain abdomen is first thought to be due to development of PVT. Moreover, our patient was haemodynamically stable, having no features of haemorrhagic shock.

5. Conclusion

Hepatitis B has many presentations varying from inactive carrier stage in majority to cirrhosis and H.C.C. The development of acute pain abdomen in background of cirrhosis should not always be attributed to PVT but tumour rupture should also be kept in mind.

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