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Case Report

HEPATOCELLULAR CARCINOMA PRESENTING AS RENAL VEIN THROMBOSIS

***Anand V Kulkarni[1], Pramod Kumar DA[1], Mithun Sharma[1], D Nageshwar Reddy[1] and Padaki Nagaraj Rao[1]**
Department of Department of Hepatology & Liver Transplant, Asian Institute of Gastroenterology, Hyderabad, India
**Author for Correspondence: anandvk90@gmail.com*

ABSTRACT
Hepatitis B is a leading cause of hepatocellular carcinoma (HCC). HCC is known to invade portal vein and hepatic veins. HCC involving the renal vein has not been reported in the literature. Here we report a rare case of Hepatitis B related hepatocellular carcinoma presenting as renal vein thrombosis.

Keywords*: Hepatic venous outflow tract obstruction; Hepatocellular carcinoma; Renal Vein thrombosis, Hepatitis B*

INTRODUCTION
Hepatitis B is a leading cause of hepatocellular carcinoma (HCC) (But *et al.*, 2008). HCC is the most common primary liver malignancy. HCC is most often asymptomatic; hence surveillance is strongly recommended in cirrhotics to detect early HCC, allowing an increase of patients suitable for curative treatment (Waly Raphael *et al.*, 2012). HCC presenting as renal vein thrombosis (RVT) has not been reported in the literature and ours is the first to report the same.

CASE
Forty-four-year-old male with history of herpes zoster (over T10 dermatome) presented with complaints of hematuria and left flank pain for 3 days. He also complained of visible veins over the back and abdomen for 20 days. On examination, the patient had pedal edema and a palpable non-tender liver 6 cms below the right costal margin with marked tenderness in the left flank. Scarring due to previous Herpes zoster (figure 1A and 1B), prominent veins with flow below upwards was noted on abdomen and back (figure 1A and 1B). Investigations revealed hemoglobin of 9.8g/dl (normal- 12-16g/dl), total leucocyte counts of 11,900 cells/mm3(normal- 4,000-11,000 cells/mm3), platelets of 73,000 cells/mm3(normal-1.5 to 4 lakh cells/mm3). His creatinine was 2.6mg/dl (normal-0.6-1.1mg/dl) and potassium of 7.2meq/dl (normal 3.5 to 5meq/dl). His serum bilirubin was 3.2mg/dl with direct fraction of 1.7mg/dl and serum albumin was 3.2g/dL (normal~3.5-5.5g/L) with normal transaminases. Blood gas analysis showed a pH of 7.24 with bicarbonate levels of 16.4meq/dl. Urine analysis revealed plenty of red blood cells with 2+ proteinuria. Tall tented T waves were noted on ECG hence hemodialysis was planned and viral markers were advised. Hepatitis B surface antigen was positive while hepatitis C and human immunodeficiency virus were negative. He underwent 3 sessions of dialysis for control of potassium levels. Ultrasonography of abdomen showed a large lesion in the liver and thrombosis in Inferior Vena cava (IVC) for which magnetic resonance imaging was advised. Axial section of contrast-enhanced magnetic resonance imaging showing a large infiltrative lesion in the liver (figure 2A), superficial veins (figure 2B), thrombus in IVC (figure 2A and 2B) and renal vein (figure 2B). He was diagnosed as Hepatitis B Related Hepatocellular Carcinoma Causing Hepatic Venous Outflow Tract Obstruction (HVOTO) And Left Renal Vein Thrombosis (RVT).

DISCUSSION
This patient presented with classical features of renal vein thrombosis and was managed aggressively. It was interesting to note HCC causing HVOTO and RVT simultaneously. His α-fetoprotein was 9812

IU/ml. He was started on sorafenib. However, he continued to develop recurrent refractory hyperkalemia and succumbed to multiorgan failure at day 12.

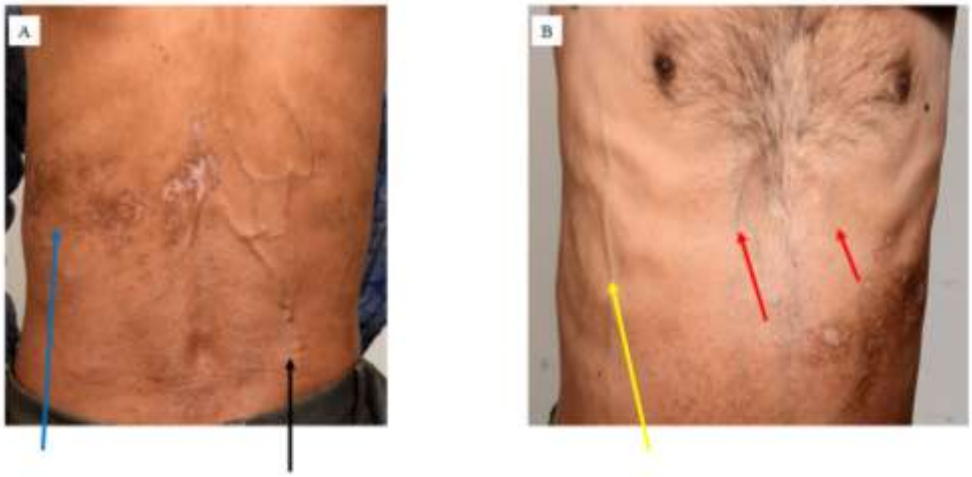
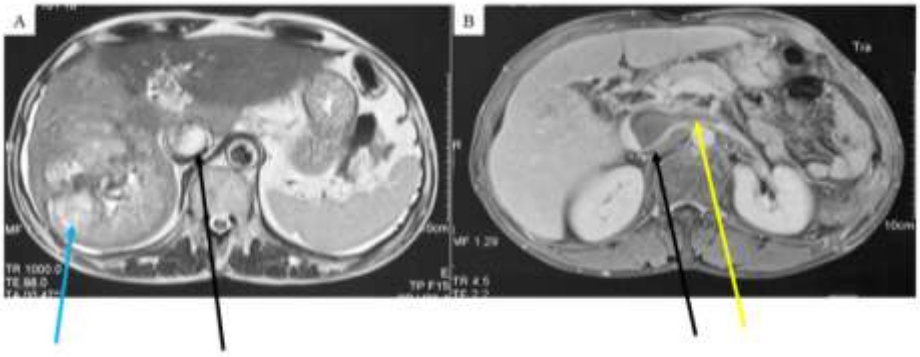


Figure 2: A- Axial section of contrast-enhanced magnetic resonance imaging showing a large infiltrative lesion in the right lobe of the liver (blue arrow in figure 2A) with thrombus in IVC (black arrow in figure 2A). B-Lower section of magnetic resonance imaging showing thrombus in IVC (black arrow in figure 2B) and renal vein (yellow arrow in figure 2B).

Hepatic venous outflow tract obstruction is usually due to Myeloproliferative Neoplasms or thrombophilic disorders (DeLeve *et al.,* 2009). Hepatocellular carcinoma (HCC) invading portal vein is quite common however HCC involving IVC as well as the renal vein is not reported in the literature (Connolly *et al.,* 2008). HCC develops in around 4% at 5 years in Budd-Chiari syndrome (BCS) especially in IVC thrombosis but HCC involving systemic veins is seen in <6% (Connolly et al.,2008; Moucari *et al.,* 2008). Malignancy associated thrombosis is multifactorial. It can be due to direct invasion of vessel or release of prothrombotic factors specially cancer procoagulant which directly activates Factor X (Hernández-Gea *et al.,* 2019).



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