***Case Report:***

# Acute viral hepatitis B complicated by acute pancreatitis.

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**Abstract:**
Majority of acute viral hepatitis cases resolve spontaneously in due course of time. Few cases that develop acute liver failure are prone to develop various complications, including acute pancreatitis. However, development of acute pancreatitis in patients with uncomplicated acute viral hepatitis is very rare and extremely rare in case of Acute Hepatitis B. In this case, a young boy with acute non-fulminant hepatitis B developed acute moderately severe pancreatitis, diagnosed by clinical, enzymatic and radiological features. He was managed conservatively and improved. Acute pancreatitis must be considered in patients with acute viral hepatitis who develop abdominal pain.

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**Introduction:**
Viral infection by hepatotropic viruses results in acute viral hepatitis, uncomplicated in most cases. Rarely, it is complicated by development of liver failure and fulminant hepatitis, in which various metabolic complications can ensue, including acute pancreatitis. However, development of acute pancreatitis (AP) in the setting of mere acute viral hepatitis (AVH) is very rare. Still rarer is the occurrence of AP in HBV related AVH.

**Case:**
19 years old male with no significant past history was admitted with fever, fatigue, anorexia, jaundice and dark urine for 5 days duration. He denied alcohol intake, prior history of gall stones, diabetes or dyslipidemia. He was fully conscious and there was no asterixis. LFT was consistent with acute viral hepatitis (Total Bilirubin = 10.5 mg/dL; ALT= 3660 IU/L; AST= 3280 IU/L; ALP= 838 IU/L) and INR was 1.38. HBsAg was positive by ELISA. IgM Anti-HAV and HEV were negative. Ultrasound abdomen ruled out obstructive jaundice.

On third day of admission, he developed severe upper abdominal pain, which radiated to back and increased with movement. Anti-spasmodic, proton pump inhibitor and Tramadol could not completely relieve his pain. Serum amylase was ordered and value was 1300 IU/L [30-165 IU/L]. Contrast Enhanced CT abdomen reported enlarged pancreas with no evidence of pancreatic necrosis with peri-pancreatic fat stranding and fluid collection, consistent with acute pancreatitis (Modified CT severity index 6) (Figure 1,2).Patient did not develop any organ failure and early enteral feeding was initiated. His condition improved over 7 days and he was discharged in stable condition. In follow up after one month, patient was absolutely symptomless with no icterus, and after 6 months, HBsAg became negative.

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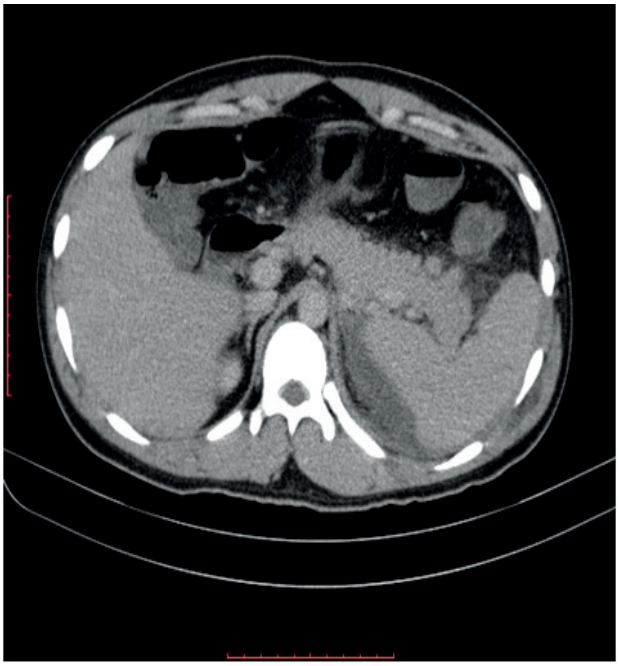

Figure 1. Cross sectional image at the level of pancreas, showing bulky tail of pancreas with peri-pancreatic fat stranding.

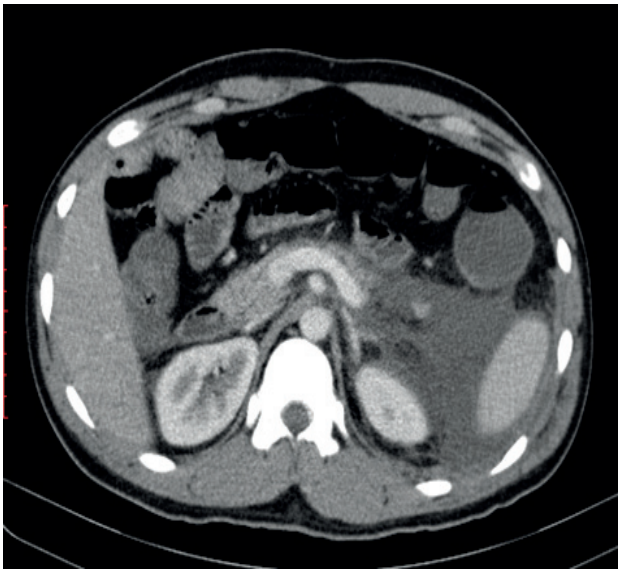

Figure 2. Cross sectional CECT abdomen image showing peri-pancreatic fluid collection with thickening of latero-conal and Gerota's fascia.

**Discussion:**

Mumps, Coxsackie virus, hepatitis B, cytomegalovirus, varicella-zoster, herpes simplex are known to be associated with AP [1]. Most cases of acute pancreatitis related to hepatitis viruses had been reported in association with acute liver failure[2]. In one series, 44% of patients who died of acute liver failure had acute pancreatitis [3]. Tissue hypoxia and hypo perfusion are proposed to be the mechanism of pathogenesis. Symptomatic pancreatitis in the setting of acute viral hepatitis are reported only in few case reports [2,4,5]. And such AP in association with acute non-fulminant Hepatitis had been reported rarely in the past. In a small case series by Jain P et al, only one patient had HBV out of total 7 cases of AP. The other one is a case report by de Oleveira SC et al, in which AP was diagnosed on typical pain and enzyme level, without CT scan[5]. Association with chronic hepatitis B is well documented on the other hand.

The exact pathogenesis of pancreatitis in these patients is not known. Edema of the ampulla of Vater with obstruction to the outflow of pancreatic fluid was proposed by Tsui CY et al[6]. Other possibility include direct inflammation and destruction of pancreatic acinar cells by the virus, which is supported by the finding of hepatitis B virus antigens in pancreatic juice aspirates of acute and chronic hepatitis B patients [7]. In conclusion, any patient with simple viral hepatitis complaining of severe epigastric pain should be evaluated for acute pancreatitis.

**Conflict of interest**: none

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