# Case Report

# Viral Hepatitis: HBV-HCV Co-infection

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## Abstract

*Co-infection with HBV and HCV is a complex clinical existence which estimated prevalence is reported 0.7% to 16% worldwide. HCV superinfection is very common due to viral replication in HCV is more dominant over HBV. Most of the clinical studies reported that disease progression is faster in HBV and HCV co-infected patients in compare to those with mono-infection. Therefore, early diagnosis and proper treatment is important for withholding the disease progression. Here a case of 45 years old male with fever, anorexia, vomiting and mucus mixed stool. HBsAg and anti-HCV are positive. USG of whole abdomen suggesting chronic liver disease with chronic kidney disease. Endoscopy of upper GIT revealed grade III esophageal varices. There are no established guidelines for treatment of HBV-HCV co-infection. Only symptomatic treatment was given.*

***Key words:*** *Hepatitis B virus, Hepatitis C virus, Co-infection, Esophageal varices*

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## Introduction

Hepatitis B (HBV) and Hepatis C (HCV) virus infection account for the leading cause of death globally. World Health Organization estimates that approximately more than 250 million and 170 million people are infected with HBV and HCV respectively.[1] In Bangladesh the prevalence of HBV is 5.4% in general population. On the other hand, there is limited data about the prevalence HCV that is estimated approximately 0.84%.[2] The mode of transmission of these two viruses are treatment from quacks by reusing of unsterilized syringes and other implements, shaving and hair trimming in barber shops, body piercing, vaccination against small pox, cholera, dental procedure, intravenous infusion and drug abusers etc.[2] Because of the shared modes of transmission coinfection with two viruses is common. HBV-HCV coinfection is more complex compare to mono-infection with HBV or HCV alone. The exact prevalence of HBV and HCV coinfection is unknown but is reported to be between 0.7% and 16% in high endemic region and among people at high risk for parenteral infection.[3-5] HCV-HBV coinfection may also occur by superinfection. HCV superinfection is more common whereas HBV superinfection is rare.[6-7]

## Case Presentation

A 45-year-old male, taxi driver was admitted to Khwaja Yunus Ali Medical College Hospital, a tertiary-level hospital in the northern part of Bangladesh owing to the passage of stool mixed with mucus 7-8 times per day for 3 days. He also complained of fever, anorexia, nausea and vomiting for same duration. On the day of hospital admission, he was reported marked jaundice with respiratory distress. The patient's drug history, he had had some herbal medicine for few days. The patient having a history of staying abroad as an expatriate for 18 years.

On admission to the hospital, the patient was alert, with a blood pressure of 110/80 mm Hg, a pulse rate of 95 bpm, a body temperature of 98°F and SPO2 92%. The bilateral palpebral conjunctiva was significantly jaundiced. Mild leg edema was present and bowel sound was normal. There was mild upper abdominal tenderness and splenomegaly. Other physical examinations revealed normal. He was initially diagnosed with acute viral hepatitis.

To determine the cause of acute hepatitis, viral antibodies, and hepatitis B surface antigen were tested. HBsAg and anti-HCV were positive. The relevant laboratory tests on admission to the hospital showed the following: ESR 37 mm in 1st hour; hemoglobin 12 g/dL; total white blood cells, $7.51\times10^9$/L with evidence of lymphocytopenia (14%); platelets, $110\times10^9$/L; prothrombin time with INR (International normalized ratio) 20.10 sec; 1.66. Serum albumin 25.88 g/L. Liver enzyme ALT level was elevated 458 U/L; but alkaline phosphatase was 81

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U/L; total bilirubin was 672.69 µmol/L; serum creatine level was 267.37µmol/L; serum electrolytes revealed hyponatremia (serum sodium 129 mmol/L). Ultrasonography of whole abdomen showed chronic liver disease (there was no biliary obstructions or mass lesions in the liver) with mild ascites, Acute kidney injury (AKI) on chronic kidney disease (CKD), bilateral renal cortical cysts and mild splenomegaly. Endoscopy of upper gastrointestinal tract (GIT) revealed esophageal varices grade III with congestive gastropathy (Figure: 1)

Hence the final diagnosis was chronic liver disease caused by hepatitis B and C virus associated with CKD and esophageal varices.The patient received only symptomatic treatment. The patient's symptoms subsided gradually, and he was discharged from the hospital.

prevalence of one virus over the other.[8] Diagnosis of acute hepatitis is difficult due to most cases are asymptomatic. Others have only nonspecific symptoms like fever, fatigue, myalgia and anorexia may misdiagnosed acute hepatitis as a common cold. When the symptoms persist more than 10 days following jaundice acute hepatitis should be considered and liver enzymes should be estimated. Jaundice is the most specific liver related symptom and around 50% to 84% symptomatic patients having the jaundice. If the liver enzymes are elevated the cause of hepatitis should be determined.[9]

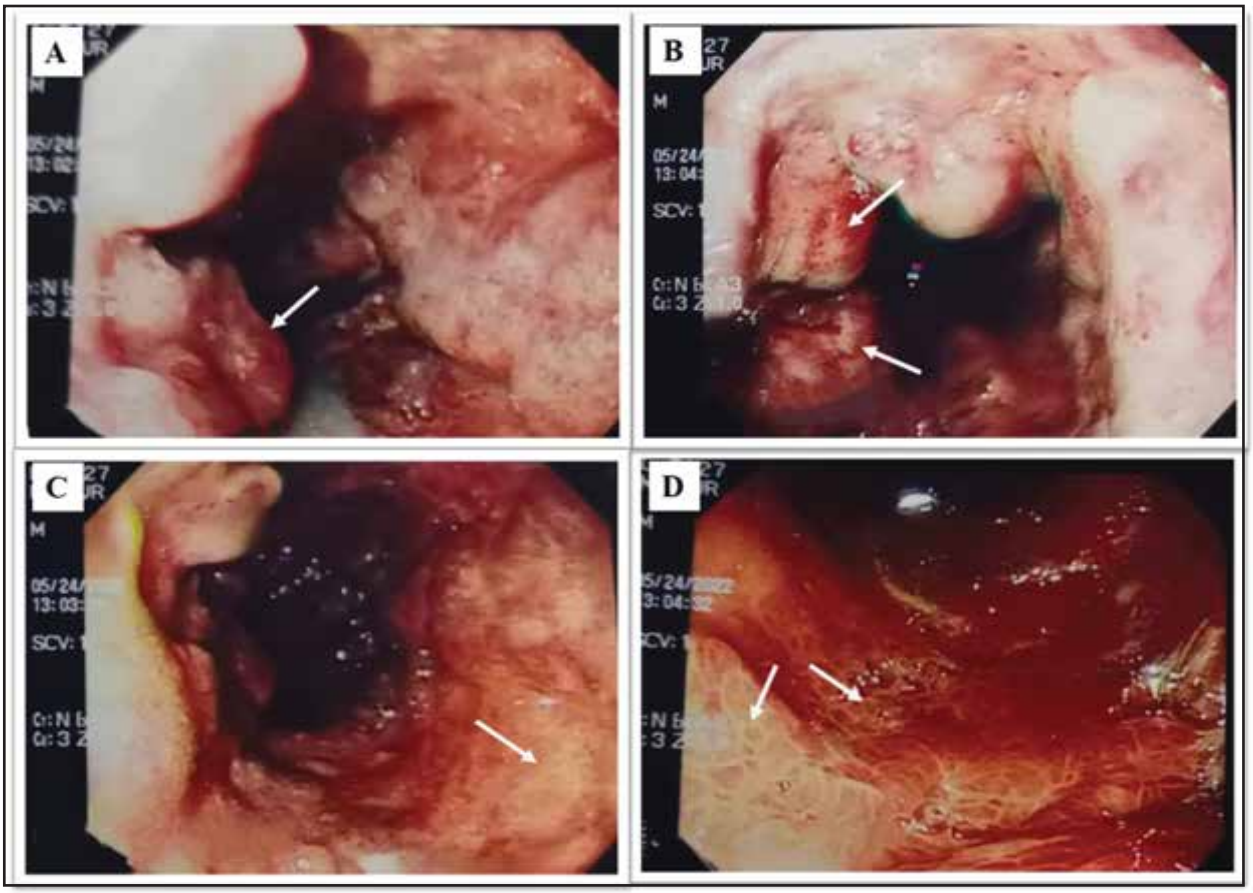


**Figure 1:** Endoscopy of the upper GIT showing (A and B) elongated dilated veins in the middle and lower 3rd of the esophagus and (C and D) multiple submucosal hemorrhagic lesions with mosaic in appearance in the fundus and body of the stomach.

## Discussion

The global prevalence of HBV and HCV coinfection has been reported 0.7% to 16% worldwide and likely to be underestimated while the disease outcomes are more severe in comparison to patients with single hepatitis virus infection. Coinfection is frequently observed in population with high-risk parenterally acquired infections. Unfortunately, patients with HBV-HCV coinfection have heterogenous clinical manifestations. Either there could be HCV predominance or HBV predominance. It indicates that acquisition time of each infection is crucial for the

Several studies have shown that HBV-HCV co-infection is a factor which prone to the progression of the liver fibrosis and the increased incidence of cirrhosis. Moreover, the coinfection with these two infections may be associated with the development of liver cancer,[10] and the risk of development of liver cancer is greatly higher in HBsAg/HCV positive cirrhotic patient than individuals infected with HCV or HBV alone. HCV has been associated with microalbuminuria. This viral infection may have higher risk and development of CKD within short time. Studies revealed that HCV infection is associated up to

2.2-folds higher mortality, rapid progression of CKD to end stage renal disease.[11] Various authors also investigated association of HBV and renal disease. HBV related membranous nephropathy is the commonest type among Asians.[12]

The incidence of esophageal varices in HBV cirrhotic patient as high as 90% and one third of cirrhotic patient with esophageal varices develop episode of esophageal hemorrhage. Mahmoud Abdel-Aty et al revealed that high incidence of HCV induced esophageal varies in Egypt.[13]

## Conclusion

Our patient developed chronic hepatitis with CKD and esophageal varices due to dual infection of HBV and HCV. The source of infection is not so clear, may have been community acquired or hospital acquired. Many patients are unaware of their infection until it becomes chronic and symptomatic. Most cases are asymptomatic or have only mild nonspecific symptoms. When infection with HBV and HCV left untreated this chronic infection contribute to decompensated cirrhosis, hepatocellular carcinoma and subsequent complications. Therefore, diagnosis and proper treatment at an early stage is of great importance for impeding disease progression.

## References


1. Peeling RW, Boeras DI, Marinucci F, Easterbrook P. The future of viral hepatitis testing: innovations in testing technologies and approaches. BMC Infect Dis 2017; 17:699.

2. Al-Mahtab M. Past, Present, and Future of Viral Hepatitis in Bangladesh. Euroasian J Hepato-Gastroenterol 2016;6(1):43-44.

3. Chakravarti A, Verma V, Jain M, Kar P. Characteristics of dual infection of hepatitis B and C viruses among patients with chronic liver disease: a study from tertiary care hospital. Trop Gastroenterol 2005; 26:183-187.

4. Mekky MA, Nasr AM, Saleh MA, et al. Virologic and histologic characterisation of dual hepatitis B and C co-infection in Egyptian patients. Arab J Gastroenterol 2013; 14:143-147.

5. Gaeta GB, Stornaiuolo G, Precone DF, et al. Epidemiological and clinical burden of chronic hepatitis B virus/hepatitis C virus infection. A multicenter Italian study. J Hepatol 2003; 39:1036-1041.

6. Liaw YF. Hepatitis C virus superinfection in patients with chronic hepatitis B virus infection. J Gastroenterol 2002;37 Suppl 13:65-68.

7. Liaw YF, Yeh CT, Tsai SL. Impact of acute hepatitis B virus superinfection on chronic hepatitis C virus infection. Am J Gastroenterol 2000; 95:2978-2980.

8. Liu CJ, Liou JM, Chen DS, Chen PJ. Natural course and treatment of dual hepatitis B virus and hepatitis C virus infections. J Formos Med Assoc 2005; 104:783-791.

9. Takamizawa S, Yamada T, Kitamura K, et al. Difficulty of acute hepatitis C diagnosis in a hospitalised patient. BMJ Case Rep 2018;11: e226907.

10. Raimondo, G., G. Cacciamo, and C. Saitta, Hepatitis B virus and hepatitis C virus co-infection: additive players in chronic liver disease? Ann Hepatol, 2005; 4(2): 100-106.

11. Khan, M.U., M.I. Mahmoud, and A.A. Butt, Hepatitis c virus and chronic kidney disease. Expert Rev Gastroenterol Hepatol, 2020; 14(7): 579-590.

12. Zeng C, Chen H, Wang R, Chen Y, Zhang S, Liu L, Li L, et al. Aetiology and clinical characteristics of membranous nephropathy in Chinese patients. Am J Kidney Dis 2008; 52: 691-698.

13. Abdel-Aty, M., et al., Incidence of HCV induced-Esophageal varices in Egypt: Valuable knowledge using data mining analysis. Medicine (Baltimore), 2017; 96(4): e5647.