

An Atypical Case Report on Hepatitis B Induced Liver Cirrhosis in 51-Year-Old Male Patient

B.Pradeepthi¹, Dr. J Bhargava Narendra*

Address: 1.V Pharm d student, Aditya Pharmacy College, Surampalem, Kakinada.

*Corresponding author: Dr.J Bhargava Narendra, Asst.Professor, Aditya Pharmacy College, Surampalem, Kakinada.

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ABSTRACT:

Hepatitis B is a life-threatening infection of liver caused by HBV. Cirrhosis and hepatocellular carcinoma are the sequelae's of the chronic hepatitis infection which are caused by either undiagnosed condition or the non-adherence of the patient to medication. Here we present a case of 51-year-old male patient with chief complaints of vomiting's, diarrhoea and itching of body for 1 week. Laboratory tests revealed increase in the liver function tests, USG abdomen showed cirrhosis and PCR test showed positive Hepatitis B core antibody (18.89 S/CO). Patient was diagnosed with Hepatitis B, 7 months back. Due to the negligence of the patient by not administering the prescribed medication the advancement of the Hepatitis B led to development of cirrhosis. The patient was prescribed with entecavir 1 mg/day which improved the symptoms of the patient during his hospital stay which improved the symptoms. Patient has more chances of developing HCC if the patient is continued to be non-adherent to the medication. In order to avoid the progression of the illness, the patient was counselled about the disease and non-pharmacological therapies, and a follow-up was conducted to ensure that the patient was administering the medication.

KEY WORDS: Hepatitis B, Cirrhosis, HCC, Portal hypertension, UTI, medication adherence

I. INTRODUCTION

Hepatitis B is a serious liver infection which is most frequently occurring viral infection in the humans that even can develop into CLD, cirrhosis and hepatocellular carcinoma. ^[1]This infection is caused by the Hepatitis B virus belongs to Hepadnaviridae family which is partially double stranded DNA virus. ^[2]Infection spreads by bodily fluids including blood, sperm, and vaginal secretions. The majority of immunocompetent persons with HBV infection are capable of recovering well on their own. ^[3]

Hepatocellular carcinoma and liver cirrhosis are the two deadly outcomes of chronic hepatitis. ^[4] Cirrhotic patients are at risk of developing various infections caused by bacteria this occurs due to compromised immune system as most of the immune proteins are produced by the liver and splenomegaly leads to reduced leucocyte production which is opportunistic for bacterial invasion. Bacterial infections include are SBP, UTI, pneumonia and Sepsis. ^[5]

Hepatocellular carcinoma is a malignancy that affects the liver. The major risk factor for developing HCC is untreated or undetected Chronic Hepatitis B. Taking appropriate preventive measures and administering the prescribed medications can prevent the development of HCC. ^[6]

Hepatitis B affects over 296 million people worldwide and is thought to cause 820000 deaths annually, according to the research from the Centre for Disease Control and Prevention. Among these, 25% of chronic hepatitis B infections advance into hepatocellular cancer. Despite having a higher chance of developing liver disease, the majority of people with hepatitis B virus infection do not go on to develop cirrhosis or hepatocellular carcinoma, making the condition uncommon based on prevalence. ^[7]

II. CASE REPORT

A 51-year-old male patient was admitted in general medicine ward with chief complaints of vomiting's, diarrhoea and itching of body for 1 week. Patient past medical history include hepatitis B for 7 months and diabetes type II for 3 years. Patient was prescribed with Tenofovir 25 mg OD, Ursodeoxycholic acid 60 mg BD for hepatitis B and metformin & glimepiride 500 mg BD for diabetes mellitus. Patient was not adherent to the medication and completed neglected the administration of the medication. Patient had abnormal bowel and bladder sounds, ascites and icterus were observed. Patient blood pressure was

119/64 mm/hg, pulse rate was 85/bpm, respiratory rate was 20/min, temperature was 99°F, blood sugar levels were 146 mg/dl. Ultra sonography of abdomen report revealed that liver has coarse echotexture with nodular surface, spleen is mildly

prominent 12mm, portal vein is enlarged in size 13 cm, minimal ascites and peripancreatic collaterals. PCR test report revealed Hepatitis B core antibody 18.89 S/CO and Hepatitis viral load 414159660viral copies/ml.

Table 01 – laboratory investigations

PARAMETERS	RESULT	INDICATION
LIVER FUNCTION TESTS		
Total bilirubin	21.3 mg/dl	Indication of hepatocellular damage
Direct	18.8 mg/dl	
Indirect	2.5 mg/dl	
AST or SGOT	825 U/L	Indicates cirrhosis and hepatocellular damage
ALT or SGPT	333 U/L	
ALP	130 IU/L	Indication of hepatocellular damage
HAEMATOLOGICAL TESTS		
Haemoglobin	12 gm%	Indicates anaemia
Total RBC count	3.96 mil/cumm	
PCV	30.9 vol%	
MCHC	38.7%	Indicates anaemia often caused by iron deficiency
Neutrophils	85%	Indicates infection and inflammation
Lymphocytes	13%	
Prothrombin time	19 sec	Indicates severe liver disease and lack of clotting factors
PLASMA PROTEINS		
Albumin	2.3 g/dl	Indicates severe cirrhosis and hepatitis infection
Globulin	4.4 g/dl	Sign of hepatic damage
RENAL FUNCTION TESTS		
Blood urea nitrogen	103 mg/dl	Indicates kidney related disorder
Serum creatinine	3.5 mg/dl	
URINE EXAMINATION		
Pus cells	4-6/HPF	Sign of urinary tract infection
Bacteria	Present	
Urine spot protein	40 mg/dl	Sign of kidney related disorder (diabetic nephropathy)
ELECTROLYTES		
Sodium	131 mmol/l	Indicates kidney disease and cirrhosis
Potassium	5.4 mmol/l	Indicates kidney disease

Chloride	106 mmol/l	
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Table 02 –

GENERIC NAME	INDICATION	DOSE	ROA	FREQUENCY
Ceftriaxone and tazobactam	Prevent Urinary Tract Infection	1.5g	IV	BD
Pantoprazole	To reduce stomach acidity	40mg	IV	OD
Ondansetron	To treat Vomiting	8mg	IV	TID
Racecadotril	To treat Diarrhoea	10 mg	PO	TID
Ursodeoxycholic acid	Hepatoprotective	60 mg	PO	BD
fortified micronutrients	To improve Nutritional status	1 tab	PO	OD
Entecavir	To treat Hepatitis B	1 mg	PO	OD
Albumin	To improve Hypoalbuminemia	20%	IV	-
Saccharomyces boulardii	Restore normal flora	1 tab	PO	BD

III. DISCUSSION

In our case study, patient has various complications which are developed due to Chronic hepatitis. Continuous damage to hepatocytes by virus led to the scarring of the liver which impaired the regulation of blood flow through the portal vein and caused portal hypertension. Several studies have indicated that hepatitis B-related cirrhosis and associated consequences are caused by T lymphocytes, neutrophil infiltration, and natural killer cells. By activating stellate cells through NF-KB, the inflammation causes oxidative stress, which in turn causes fibrosis and ultimately cirrhosis. The immune cells are proactively targeting the virus and causing damage the hepatocytes at the same time. Cirrhosis of the liver is caused by an ongoing infection-induced inflammatory-necrotic-regenerative process. The main immune cells that harm the liver are CD8+ T lymphocytes and natural killer cells. By attacking the hepatocytes directly and causing the release of pro-inflammatory chemical mediators, the hepatic cells secrete fibrosis-related substances that lead to liver fibrosis and ultimately cirrhosis. The buildup of extracellular matrix proteins results in fibrosis, which also happens as a result of the tissue healing that follows inflammation.^[8,9] Yan Chao Zhou, Yulin wan et al described a hypothesis regarding

the advancement of hepatitis B to cirrhosis and liver cancer. The hypothesis proposes that Collagen expression in the liver is triggered by the HBV X protein's ability to capture protons and chlorides. 20% of collagen has proline and hydroxyproline, which may create strong hydrogen bonds with entrapped protons. From liver infections to fibrosis, the development of collagen is an important phase. Some people with liver cirrhosis develop liver cancer because the X protein and collagen work together to accumulate HCl locally.^[10] According to Yasuko Iwakiri et al, the increased pressure in the portal vein develops due to the increased hepatic vascular resistance due to dysregulation of the liver sinusoidal endothelial cells, stellate cells and inflamed infiltrated macrophages because of the persistent hepatitis B and cirrhosis. This leads to extra hepatic vasculature changes which aggravates the portal hypertension.^[11]

Patient urine examination revealed UTI which is a major complication of the cirrhosis and urine spot protein is also identified this indicates the diabetic nephropathy. Patient has been prescribed with ceftriaxone which is a primary drug for treating the UTI. While pneumonia, cellulitis, and bacteraemia are relatively rare, spontaneous bacterial peritonitis (SBP) and urinary tract infection (UTI) are the most prevalent conditions. Multiple pathophysiological pathways, including

gut dysbiosis, enhanced bacterial translocation, portosystemic shunting, cirrhosis-associated immune dysfunction (CAID), liver failure, and hereditary variables all contribute to infections in these individuals. Prevention, early detection, and effective treatment of infections may result in a reduction in morbidity and death. The preferred treatment for urinary tract infections caused on by cirrhosis is intravenous third-generation cephalosporins or piperacillin/tazobactam.^[12] According to Samuel N. Heyman et.al diabetes is one of the causes of the kidney related diseases and proteinuria/urine spot protein and albuminuria are the hall mark for diabetic nephropathy which is developed in both type I and type II diabetes patients.^[13]

Patient liver function test revealed abnormal bilirubin, AST, ALT and ALP levels. According to B.R. Thapa et.al liver execute number of functions such as biochemical, synthetic and detoxifying processes. Liver function tests are one of the evaluating tools for detecting liver diseases.^[14]

Patient was prescribed with Entecavir 1mg/day which improved the symptoms with continuous administration of the drug. The anti-viral agents are to be administered continuously when the hepatitis has been diagnosed which prevents the advancement in the hepatitis condition to cirrhosis or furthermore to hepatocellular carcinoma. Patient have high chances of development of hepatocellular carcinoma based on his lab reports. Chang et.al, has conducted a study on Nucleoside-naive, HBeAg⁽⁺⁾ and HBeAg⁽⁻⁾ patients for 3 years. The study concluded that chronic hepatitis B with advanced cirrhosis or fibrosis can be treated or reversed with the long-term treatment with entecavir.^[15]

IV. CONCLUSION

The patient has improved his symptoms after administrating entecavir continuously for one week along with Ursodeoxycholic acid, racecadotril and ondansetron. Reduction in the values of liver function test had been observed within 7 days of treatment. Regular administration of medication necessary to prevent further complications. Medication adherence of the patient and persistent follow up is the only key to improve the patient quality of life. As a Clinical Pharmacists, we have provided appropriate counselling regarding the disease, medication and life style modification, for both the patient as well

as care givers to avoid the previous errors and to improve the patient condition.

Hepatitis is a serious condition which require immediate therapeutic interventions to prevent all the complications caused by the HBV. Similarly patient counselling also plays a huge role in educating the patient regarding his condition and necessity of administering the medication. It also improves the medication compliance of the patient. Here the treatment given to the patient was rational and aimed to reverse the condition and improve the patient quality of life.

ABBREVIATIONS:

SBP: Spontaneous Bacterial Peritonitis, **PCR test:** polymerase chain reaction test, **HBV:** Hepatitis B virus, **HCC:** Hepatocellular carcinoma, **OD:** Once 1 a day, **BD:** twice in a day, **AST:** Aspartate Transaminase, **ALT:** Alanine Transaminase, **SGOT:** Glutamic oxaloacetic transaminase, **SGPT:** Serum Glutamate Pyruvate Transaminase, **ALP:** Alkaline Phosphatase, **RBC:** Red Blood Cells, **PCV:** Packed Cell Volume, **MCHC:** Mean Corpuscular Haemoglobin Concentration, **IV:** Intravenous, **TID:** Thrice in a day, **PO:** Per oral, **NF-kB:** Nuclear transcriptional activator that binds to enhancer elements in many different cell types

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CONFLICT OF INTEREST:

The authors declare that there is no conflict of interest

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