ISSN 2091-2889 (Online)
ISSN 2091-2412 (Paper)

Journal of Chitwan Medical College 2013, 3(3): 65-66
Available online at: www.jcmc.cmc.edu.np

CASE REPORT

CLINICAL STATUS OF FUROSEMIDE ON LIVER CIRRHOSIS WITH PORTAL HYPERTENSION AND ASCITES

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ABSTRACT

Cirrhosis of liver is a pathologically defined entity that is associated with a spectrum of characteristic clinical manifestations. Portal hypertension is one of the complication of cirrhosis of liver. We report the case of a 55-year-old man with liver cirrhosis and subsequently portal hypertension and ascites who was admitted to the hospital with headache and indigestion. The patient was treated with furosemide 40 mg OD, lactulose 10 ml HS, Tone, vit K 10 gm IV, Usoliv 300 mg BD and pantoprazole 40 mg OD. The relevant and evidence based clinical practice guidelines and clinical status of furosemide on particular condition were discussed with treating physician.

Key Words: *Liver Cirrhosis, portal hypertension, ascites & furosemide*

INTRODUCTION

Cirrhosis of the liver refers to scarring of the liver which results in abnormal liver function as a consequence of chronic (long-term) liver injury. Cirrhosis occurs when the normal structure of the liver is disrupted by bands of scar tissue. One of the normal functions of the liver is to filter blood returning to the heart from the digestive system. When cirrhosis is present, the presence of scar tissue causes increased resistance to blood flow through the liver. This result in high pressures developing in the veins that drain into the liver, a process called portal hypertension. Many of the complications of liver disease, such as fluid retention and esophageal bleeding, are caused by the presence of portal hypertension. Portal hypertension can lead to life-threatening hemorrhage, ascites, and encephalopathy. [1] Management of portal hypertension and its attendant complications requires a multidisciplinary approach combining medical and endoscopic management, surgical or nonsurgical portosystemic shunting, and in some cases liver transplantation. [2] We present a case of liver cirrhosis with portal hypertension which could have resulted due to regular intake of alcohol as per patient's history. The pharmacokinetic and pharmacodynamic aspects of furosemide regarding particular disease were discussed with the treating physician.

CASE PRESENTATION

A 55 years old male patient was admitted to the hospital complaining of headache and indigestion for 15-20 days. The patient on examination showed yellowish discoloration of sclera and yellowish discoloration of tongue. There was no significant medical history. As per the patient, he is alcoholic and use to consume 4-5 quarter of alcohol/day (in patient's language). An advanced CECT abdomen was performed and CT scan report was suggestive of F/S/O cirrhosis of liver with portal hypertension, distended GB with sludge, mild splenomegaly, and mild to moderate ascites. The lab values for LFT were above the normal range (Bilirubin: 17.0 mg/dl, Bilirubin direct: 13.0, SGPT 65.0 IU/L, SGOT: 190, Alkaline Phosphatase: 1126). UGI endoscopy report showed grade II-III esophageal varices. ADA test was performed whose report was found to be within normal limit. Exfoliative cytology test for malignancy was negative. After completing required diagnostic procedure and clinical examinations, the physician started treatment with following drugs; T. Pantoprazole 40 mg OD, T. Usoliv 300 mg BD, T. Tone 100 mg OD, T. Furosemide 40 mg OD, Syp Lactulose 10 ml HS, Inj. Vit K 10 mg IV.

DISCUSSION

The diagnosis of cirrhosis of liver with portal hypertension was based on clinical status and abnormalities in lab tests. To prevent hepatic encephalopathy and minimizing the portal hypertension were the first objectives of treating physician. The hyperdynamic circulation begins in portal venous bed as a consequence of portal hypertension due to increased resistance to flow from altered hepatic vascular morphology of the chronic liver disease. [3] Alcoholism is a major public health problem and resembles,

in many ways, other chronic relapsing medical conditions. [4] The use of furosemide was mainly targeted for reducing ascites. As far as clinical status of furosemide is concerned, only minimal changes in pharmacokinetic parameters and marginal alteration in the plasma concentration-time curve was determined when the researchers studied various factors that influence intersubject variability in response to furosemide. Furosemide's pharmacokinetics were not, therefore, appreciably altered by cirrhosis. However, cirrhosis was associated with a reduction in pharmacodynamic response to this diuretic. [5] Yet another study done on patients with chronic liver disease and ascites, it was found that the furosemide kinetic patterns altered. There were increased in t1/2, Vdarea, and Vdss. The clearance of furosemide from plasma, however was not greater in cirrhotic patients than in normal. This reflects the proportionate increase in the Vd and the elimination t1/2. Portal hypertension can lead to life-threatening variceal hemorrhage or development of morbid ascites and encephalopathy. [6] Spironolactone is the first-line diuretic of choice, as it is an aldosterone antagonist and the addition of furosemide, although not confirmed in randomized trials, is thought to prevent hyperkalemia. A combination of spironolactone and furosemide improves ascites by targeting the renin-aldosterone system while preventing hypokalemia. [2] Some studies also suggests that spironolactone alone seems to be as safe and effective as spironolactone associated with furosemide, in terms of response rate and rapidity of moderate ascites mobilization in nonazotemic cirrhosis. [7] Liver transplantation is he only therapy that addresses both portal hypertension and the underlying liver disease. [2]

CONCLUSION

Finally, furosemide, is clinically found to be beneficial for patient having liver cirrhosis with portal hypertension and ascites, as the patient's symptoms were improved during discharge and the treating physician asked for a follow-up. And as the pharmacokinetic parameters of furosemide is not significantly altered in cirrhotic patient, lower diuretic efficacy of furosemide is attributed to its reduced pharmacodynamic effect. However, if the patient is not hyperkalemic, for ascites, spironolactone alone or combination with furosemide is more suggestive. Since spironolactone alone requires less dose adjustment, it would be more suitable to be used on an outpatient basis.

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