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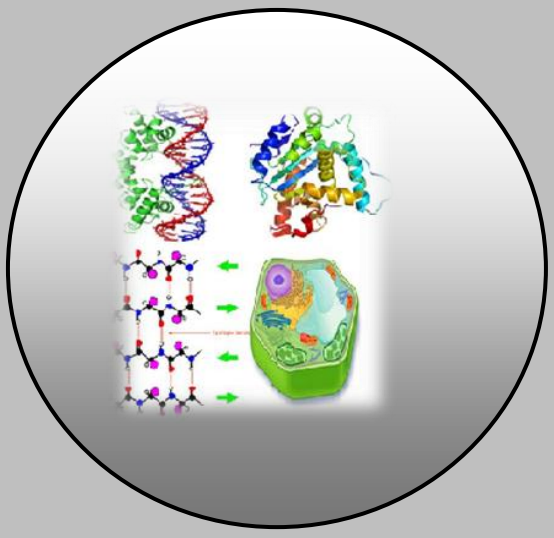

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jbiolchemres@gmail.com
info@jbcr.in

SHORT COMMUNICATION

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Liver Cirrhosis Disease: A Case Based Study

M. M. Khan, M.S. Naqvi* and M.T. Khan I. Pisharody, S. Sehgal and F. Khan*****

Department of Physiology, Career Institute of Medical Sciences and Hospital, Lucknow, U.P., India
Department of Zoology, Shia Postgraduate College, Lucknow, U.P., India*
Department of Pathology, Thomas Jefferson University, Philadelphia, U.S.A**
Department of Prosthodontics & Crown & Bridge, Kothiwal Dental College & Research Centre, Moradabad, U.P., India***

ABSTRACT

Liver cirrhosis or end-stage liver disease is a chronic disease of liver leading to irreversible fibrosis and hepatic cell destruction. The disease is manifested initially by extreme fatigue, loss of appetite and weight loss. As the liver function declines, water starts accumulating in the legs and abdomen, and the jaundice and bleeding may occur. Patients become increasingly sensitive to medications and their side effects. Apart from medication, a number of other interventions such as family support, patient's education and health care professional plan are required for the relatively successful management of such a fatal disease. Following is the case study of a patient with Liver cirrhosis.

Key words - Liver cirrhosis, Fibrosis, Jaundice, Loss of appetite and Ascitis

INTRODUCTION

Descriptor- KK is a 48 years old male with a history of alcohol cirrhosis admitted to GI unit at Postgraduate hospital.

Subjective- Patients complains of abdominal pain, unproductive cough, short of breath, weakness, malaise, poor appetite chills and sweat. He also complains of cramps in the left foot and very itchy back.

Objective-

Past medical history - Alcohol cirrhosis, non-bleeding esophageal varices, refractory ascites, DVT/PE.

Drug allergies- sulfa, penicillins

Prior medications- amiloride 30mg, 1qd, cipro, buspar, calcium carbonate, lactulose, nodalol, nicoderm, quinine-sulfate.

Current medications-

amiloride 20mg, 1qd PO for diuresis
amylase/lipase capsule, 1qd PO as enzyme supplement cipro 750 mg, 1qd PO for prophylaxis against infection furosemide 60mg, 1qd, IV for diuresis
lactulose 20g, 1qd PO for hepatic encephalapathy
Pentaprozole 40mg, 1qd PO for prophylaxis against esophageal reflux hydroxyzine 25mg, 1q6h PO PRN for itching
Maalox suspension 30ml, 1q6h PO PRN for constipation oxycodone 5mg, 1q6h IV PRN for pain
quinine sulfate 260mg, 1qhs PO PRN for cramp

Vitals-

11/22/09 BP 93/53, HR 82, RR 20, T 97.6
11/23/09 BP 89/54, HR 86, RR 18, T 99
11/25/09 BP 82/37, HR 83, RR 18, T 98.9

Physical exam- General- Patient in mild distress
Abdomen- hard, swollen with ascites, (+) bowel sound
Cardivascular- (-) S1/S2 sound
Lungs- Clear
Extremities- swollen left feet
Head and Face- Temporal wasting
Eyes- PEERL, pink conjunctiva
Neuro- confused
Height- 6 feet, Total body weight- 83.55 kg
Pathology Tests- (-) culture, no organism/no growth in two days.
Labs- 11/22/09

Na 137, K 3.3, Cl 99, CO2 32, BUN 6, SCr 0.8, Glucose 72, WBC 4.3, MCV 100, Hgb 114, Hct 33.6, Platelets 93, Lymphocytes 23.5, Eosiniphils 3.8, PT 23.3, INR 1.92, PTT 41, Alb 2.4, TB 6.5, DB 2, Alkaline phosphate 150, ALT 19, AST 61, Lipase 35,
Amylase 72.

Na 133, K 4.4, Cl 101, CO2 28, BUN 7, SCr 0.7, Glucose 80, Ca 4.4, PO4 3.2, Mg 1.3, WBC 4.2, MCV 99, Hgb 99, Hct 28.5, Platelets 77, Lymphocytes 23.2, Eosinophils 2.1, Albumin 2.1.

Social history- (+) heavy drinker, quit now
(+) substance abuser, quit
now Family history- Mother alive, afib
Father died MI 63

Patient's present condition is typical of liver cirrhosis.

Liver cirrhosis can be defined as a chronic disease of the liver with widespread hepatic parenchymal cell injury and hepatocyte destruction and their replacement with fibrous tissues (Talwalkar, J.A. and Lindor, K.D. 2003. And Prince, M.I., Chetwynd, A., and Craig, W.L. 2004.) As fibrosis replaces normal hepatic parenchyma, resistance to blood flow results in portal hypertension, and development of varices and ascites. Hepatic loss and intra-hepatic shunting of blood results in diminished metabolic and synthetic function, which leads to hepatic encephalopathy and coagulopathy (Kumagi, T. and Heatcote, J.E. and Orph, J. 2008). The most common cause of cirrhosis is viral hepatitis and alcoholism. There is no specific sign and symptoms of alcoholic liver disease. Generally, it includes anorexia, nausea, abdominal discomfort, weakness, weight loss and malaise. Clinical jaundice is often a late manifestation of cirrhosis. Upon physical exam, enlargement of liver and spleen and evidence of portal hypertension (e.g. ascites, peripheral edema, jaundice) may be noted. Spider angiomas are often observed in patients with advanced liver disease as red vascular figures on the skin. The other non-specific findings are palmar erythema, an intensed flushing of the skin over the meaty part of the palm opposite from the thumb (**4- Kaplan, M.M. and Gershwin, M.E. 2005**).

Portal hypertension (PHT) - Under normal physiological condition, the portal vein collects venous blood from the splanchic circulation (GI tract, pancreas and spleen) and transports the blood to the liver. When portal blood flow is impeded and portal pressure exceeds 12 mmHg, it is called portal hypertension.

Varices- The most clinical sequence of PHT is the development of varices or alternate route of blood flow. Varices decompress the portal venous system, return blood to the systemic circulation. Hemorrhage from varices occurs in 25%-40% of patients with cirrhosis and each episodes of bleeding carries a 30% risk of death.

Ascites- Ascites the pathological accumulation of lymph fluid within the peritoneal cavity is one of the earliest and most common complications of cirrhosis. More than 50% of cirrhotic patients develop ascites within 10 years of diagnosis of cirrhosis.

Hepatic encephalopathy (HE) - It is a complex neuropsychiatric syndrome with a broad spectrum of clinical sign and symptoms of neurological impairment. It occurs as a consequence elevated arterial ammonia levels.

Coagulation defects- Cirrhosis causes in the reduction of clotting factors I, II, V, VII, IX and X as well as reduction in the clearance of activated clotting factors. This results in the prolongation of the bleeding time.

Liver function tests such as AST, ALT, GGT, alkaline phosphate and other test including phosphate, bilirubin and albumin are often the first step in the evaluation of patients who presents with symptoms or signs of cirrhosis. Generally, ALT is specific to hepatocyte but AST is located in mitochondria and can also be found in heart, kidney and brain. Thrombocytopenia is a relatively common feature in both acute and chronic liver disease and is proportional to the extent of liver disease (Kaplan, M.M. and Gershwin, M.E. 2005).

Treatment of liver cirrhosis- There is no specific therapy for cirrhosis, but drugs commonly are used to treat the secondary complications of cirrhosis. The main treatment requires a complete abstinence from alcohol. Nutritional deficiency is common among alcoholics with liver disease (Kaplan, M.M. and Gershwin, M.E. 2005. Kaplan, M.M. 1996). A single dose of multivitamins with folic acid is sufficient to replenish the vitamin deficiency.

Primary prophylaxis for portal HT and varicieal bleeding- Use of non-selective beta-adrenergic blocking agents such as propanolol or nodalol block the adrenergic dialatory tone of the mesenteric arterioles resulting in unopposed alfa-adrenergic mediated vasoconstriction and decreases in portal pressure. Initiate therapy with oral propanolol 10mg TID or nodalol 20mg 1qd. For acute bleeding, packed RBCs, fresh frozen plasma and platelets may be given. Vasoactive drug therapy (somatostatin, octretide or terlipressin) to stop or slow bleeding is routinely employed. Give octretide as IV bolus of 50-100 micrograms followed by a continuous infusion of 25 micrograms/h. Monitor the patient for hypo and hyperglycemia. Antibiotic therapy to prevent sepsis should also be implemented early for patients with sign of infections. The development of TIPS provides a major improvement in the management of refractory or severe cases of oesophageal varicial bleeding and other complications of portal HT. The TIPS procedure involves the placement of one or more stents between the hepatic vein and the portal vein.

Ascites and spontaneous peritonitis- In addition to alcohol abstinence, primary treatment is salt restriction to 2g/day and oral diuretic therapy. The recommended diuretic therapy involves a single morning dose of spiranalactone 100mg and furosemide 40mg administered with the goal of 0.5 kg maximum daily loss. This combination usually maintains normal K levels. The dose may be increased up to a maximum of 400mg spiranolactone/160mg furosemide. If tense ascites is present, a 4-6 L parecentesis should be performed before diuretic therapy. Give 40g albumin per 4-6 L fluid withdrawn. Monitor for renal function and electrolytes. Liver transplant should be considered in patients with refractory ascites. Patients with spontaneous bacterial peritonitis should receive empiric broad spectrum antibiotic therapy. Cefotaxime 2g q8h for 5 days or a similar third generation cephalosporins are considered the drugs of choice.

Hepatic encephalopathy- In addition to protein intake limit to 10-20g/d, initiate lactulose therapy at a dose of 30-60 ml q1-2h until cataharis begins. The dose is then decreased to 15-30 ml PO qid and titrate to produce 204 soft stools per day. Monitor electrolytes periodically, follow mental status and number of stools per day. Antibiotic therapy with either metronidazole or neomycin is reserved for patients who do not respond to diet and lactulose therapy. There combination may provide additive effects and improve clinical response.

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Corresponding author: Prof. M.M. Khan, Vice Principal and Head, Department of Physiology, Career Institute of Medical Sciences and Hospital, Lucknow, U.P. India Email id: **dr.muslimaftabkhan@rediffmail.com**