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A case study on decompensated liver disease in a sexagenarian woman with portal hypertension and hepatorenal syndrome

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ABSTRACT:

Decompensated chronic liver disease is an instantaneous worsening of liver functions with cirrhosis. Presenting a clinical case of a sexagenarian woman with known complaints of type 2 diabetes mellitus and systemic hypertension with decompensated chronic liver disease and its complications like portal hypertension, and hepatorenal syndrome with laboratory parameters and treatment modalities are the specific objectives of this case study. A 65 years old female patient with a known case of type 2 diabetes mellitus and systemic hypertension was admitted to the hospital with complaints of abdominal distension and a history of bilateral pitting pedal oedema. Her abdominal girth was measured to be 120 cm. The discharge summary consisted of tablets of sodium bicarbonate 325 mg and calcium carbonate 500 mg every 8 h along with tablet rifaximin 550 mg thrice daily. Tablet propranolol 20 mg twice daily, tablet atorvastatin 20 mg every night, and tablet glipizide 5 mg every 12 h were also advised. Decompensated liver disease will mask the original fasting and post-prandial blood glucose levels. Treatment of diabetes in decompensated liver disease is difficult and close monitoring for hypoglycemia is obligatory.

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Keywords: Decompensated liver disease, hydrothorax, hepatorenal syndrome, ascites, rifaximin, lactulose.

INTRODUCTION:

Decompensated chronic liver disease is an instantaneous worsening of liver functions with cirrhosis and is clinically presented by jaundice, ascites, hepatic encephalopathy, hepatorenal syndrome or variceal haemorrhage [1]. The first hepatic decompensation event significantly increases the risk of further complications of liver cirrhosis and at last decompensation episodes will occur [2]. The fatality rate in patients with decompensated cirrhosis is ten times more than in the normal population [3]. The patients affected by compensated and decompensated cirrhosis are known to have diverse clinical outcomes [4]. The likelihood of

transitioning from a compensated to decompensated state varies from 4 to 12 % per year. Given that, majority of deaths in patients with compensated cirrhosis is due to the progression of decompensated state and its complications [5]. The progression of the decompensated state may be further accelerated by the development of other complications such as rebleeding, and acute kidney injury, with or without the features of hepatopulmonary syndrome, portopulmonary hypertension, cirrhotic cardiomyopathy and bacterial infections [6]. A hepatorenal syndrome is a form of kidney function wreckage that characteristically occurs in cirrhosis. Typically, the hepatorenal syndrome occurs in two different clinical patterns, type 1 hepatorenal syndrome is the acute impairment of kidney function and type 2 hepatorenal syndrome is the chronic impairment of kidney function [7]. The pathophysiological hallmark of hepatorenal syndrome is vasoconstriction of the renal circulation. However, the mechanism of vasoconstriction is not clearly understood; it may be a multifaceted involving disturbance in the circulatory function and activity of systemic and renal vasoactive mechanisms [8]. Presenting a clinical case of a sexagenarian woman with known complaints of type 2 diabetes mellitus and systemic hypertension with decompensated chronic liver disease and its complications like portal hypertension, and hepatorenal syndrome with laboratory parameters and treatment modalities are the specific objectives of this case study.

CASE REPORT:

A 65 years old female patient with a known case of type 2 diabetes mellitus and systemic hypertension was admitted to the general medicine department of the hospital with complaints of abdominal distension and a history of bilateral pitting pedal oedema. Her abdominal girth was measured to be 120 cm. The patient explained that she was diagnosed with diabetes for 15 years and hypertension for 11 years but is on irregular medication for both. The patient has a recent history of decompensated liver disease then she was also experiencing complications like hepatorenal syndrome and right-side hepatic hydrothorax addition. She also had complaints of breathlessness for the past two days. She was observed to be tachypneic at rest.

On investigation, blood pressure was observed to be higher than normal. She also felt drowsy. The blood report reveals that the patient was anaemic. Random blood sugar, fasting and postprandial blood sugar levels were higher than normal. Severe portal hypertensive gastropathy was observed. The vitals were given in Table 1 followed by clinical laboratory parameters in Table 2.

Table 1. Vitals of the patient.

Parameter	Observed value
Temperature	98.4-degree Fahrenheit
Pulse	92 beats/minute
Blood Pressure	150/90 mmHg
Respiratory Rate	24 cycles/min
SpO2	96%
Central Nervous System	Conscious
Cardio Vascular System	S1S2 heard
Respiratory System	Bi-Lateral air entry
Abdomen	Distended

Pleural fluid tapping was done. The level of glucose in pleural fluid was found to be 239 mg/dL. The protein level in the pleural fluid was found to be 3 g/dL. The ECG report of the patient shows normal sinus rhythm. No significant pathologies were observed in C.T.'s brain. Cytopathology investigation reveals the presence of small mature lymphocytes in a proteinaceous background. This is evidence of the condition of spontaneous bacterial perforation in this patient.

USG abdomen and pelvis scan divulge the conditions of the contracted liver, massive ascites and mild splenomegaly. The abdomen also showed shifting dullness. The left kidney was also observed to be contacted.

The patient has been given antibiotics cefotaxime 1 g in intravenous route every 12 h and metronidazole 400 mg orally every 8 h for 7 days. Then the regimen had been changed to ampicillin 1g intravenously every 12 h and Norfloxacin 400 mg orally every 12 h for the rest of the days.

Vitamin K injection was administered once a day. Lactulose 10 ml was given every 8 h. Diuretics Furosemide 60 mg has been given intravenously every 8 h along with spironolactone 100 mg via oral route every day. Propranolol 20 mg was administered orally every 12 h. Amlodipine 5 mg was administered via oral route every morning. Iron sucrose 100 mg was infused with 100 ml normal saline and over 1 h intermittently.

Table 2. Clinical laboratory investigation report.

Blood	Observed value	Normal value
Total Count	4800 cells/cu.mm	4000-11000 cells/cu.mm
Differential Count	Polymorphs - 74 % Lymphocytes - 24 % Monocytes - 2 %	Polymorphs- 40-70 % Lymphocytes- 20-40 % Monocytes- 2-6 %
Haemoglobin	7.1 g %	11-15 g %
Red Blood Count	2.1 million/cu.mm	4-6 million/cu.mm
Platelet	1.3 lakhs/cu.mm	1.5-4.5 lakhs/cu.mm
Packed Cell Volume	23 %	39-49 %
Urea	62 mg/dL	7-20 mg/dL
Creatine	2.8 mg/dL	0.1-1.0 mg/dL
Fasting Blood Sugar	184 mg/dL	70-110 mg/dL
Postprandial Blood Sugar	226 mg/dL	<140 mg/dL
Adenosine deaminase	13 IU/L	<40 IU/L
Random Blood Sugar	209 mg/dL	80-120 mg/dL
SGOT	44 IU/L	10-40 IU/L
SGPT	34 IU/L	10-40 IU/L
ALP	135 IU/L	70-130 IU/L
Serum electrolytes Sodium Potassium Chloride	 132 mEq/L 4.6 mEq/L 102 mEq/L	 135-145 mEq/L 3.6-5.5 mEq/L 96-106 mEq/L

SGOT: Serum glutamate oxaloacetate transaminase, SGPT: Serum glutamate pyruvate transaminase, and ALP: Alkaline phosphatase.

Four units of fresh frozen plasma have been transfused once during the hospital stay.

The discharge summary consisted of tablets of sodium bicarbonate 325 mg and calcium carbonate 500 mg every 8 h along with tablet rifaximin 550 mg thrice daily. Tablet propranolol 20 mg twice daily, tablet atorvastatin 20 mg every night and tablet glipizide 5 mg every 12 h were also advised.

DISCUSSION:

Decompensated chronic liver disease is distinguished by the evolution of complications related to portal hypertension, ascites, spontaneous bacterial perforation, and hepatorenal syndrome [9]. This case of 65 years old female patient also shows the manifestations of portal hypertension, ascites, spontaneous bacterial perforation and hepato renal syndrome. Along with this, the right-side hepatic hydrothorax is also present in this case. This patient had been treated with furosemide, propranolol, glipizide, rifaximin, lactulose, vitamin K, spironolactone and amlodipine. Along with this, to treat pleural effusion and spontaneous bacterial perforation cefotaxime and metronidazole were given. For the management of hepatorenal syndrome sodium bicarbonate and calcium carbonate were given.

Rifaximin is an evidence-based medicine for patients with decompensated liver disease for preventing recurrent hepatic encephalopathy. Its efficacy and safety were established by large randomized clinical trials [10]. Rifaximin is thought to destroy deaminating enteric bacteria to limit the production of nitrogenous compounds that are consequently absorbed and cause

hepatic encephalopathy [11]. Rifaximin is given to this patient at the dose of 550 mg thrice daily. Lactulose, a non-absorbable disaccharide is proven as first-line therapy for acute hepatic encephalopathy [12]. Lactulose is metabolised by colonic bacteria to fatty acids with a consequent decrease in pH in the colon. Lactulose also promotes escalated uptake of ammonia by colonic bacteria. The colonic bacteria utilise ammonia as a nitrogen source for protein synthesis. This, therefore, decreases the overall absorption of ammonia, a toxic chemical [13].

The role of vitamin K in the management of cirrhosis-related coagulopathy is still the tip of the iceberg. Routine use of vitamin K in liver cirrhosis cases without coagulopathy like bleeding should be avoided [14].

Many moderate drug-drug interactions have been found in this prescription. Drug-drug interactions are inevitable during the presence of polypharmacy and comorbidities. However, care must be taken to prevent the occurrence of drug-drug interactions to refrain from the development of further complications in this patient. Laboratory investigations like international normalised ratio, serum albumin, and bilirubin must be done. This will be feasible for the classification of the patient's condition according to the child-pugh score.

CONCLUSION:

The liver is a principal metabolizing organ and its failure results in an array of complications like ascites, hepatorenal syndrome, hepatic encephalopathy, hydrothorax and pleural effusion. Dose adjustments in liver disease are also arduous.

It is apt to say that misfortunes never come singly for the medical condition of decompensated liver disease. Long-lasting Type 2 diabetes mellitus along with hypertension is the major risk factor for the progression of compensated to decompensated liver disease. Decompensated liver disease will also mask the original fasting and post-prandial blood glucose levels. Treatment of diabetes in decompensated liver disease is difficult and close monitoring for hypoglycaemia is obligatory.

Though acarbose is the first-line agent for the treatment of type 2 diabetes in decompensated liver disease, hepatotoxicity is reported only infrequently with second-generation sulfonylureas like glipizide. Rifaximin and lactulose are well-established therapeutic options for the management of hepatic encephalopathy which may arise as a complication of decompensated liver disease.

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