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Case Report

Medical Negligence in Treating Chronic Liver Disease

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Abstract

Medical negligence is the misconduct of a medical professional or practitioner due to inadequate care. Herein, we report a case of chronic liver disease with hepatic encephalopathy, ascites, and esophageal variceal bleeding. In this case, the patient had moderate ascites with peripheral edema and increased blood pressure, he never received a diuretic though. Furthermore, this patient has a history of variceal bleeding and is at a high risk for variceal bleeding. Despite this, he wasn't on secondary prophylaxis to prevent rebleeding. Some drugs were added to the regimen of this patient even though they are unnecessary or with no indication. Therefore, we think it is necessary to revise the health system and working conditions in hospitals and to develop and stick to clinical practice guideline.

Keywords: Cirrhosis; Liver; Malpractice; Negligence

Introduction

Negligence generally defined as substandard behavior. Clinical negligence occurs when a health care professional fails to provide medical care. This is an act or omission that does not meet the expected level of reasonable care and results in preventable harm [1].

One of the biggest organs in the body is the liver. It is an important center for many physiological processes. These include endocrine control of, immune system support, blood volume regulation, macronutrient metabolism, the breakdown of xenobiotic substances including many current drugs, growth signaling pathways, lipid and cholesterol homeostasis, etc. The processing, breakdown and metabolism of macronutrients is one of the most important functions of the liver as it provides the energy required for these processes [2]. Cirrhosis is a situation in which the liver is scarred and permanently broken. Healthy liver tissues are replaced by scar tissue, which inhibits the liver from operating normally. It is a terminal image of chronic liver disease. During the progression from the compensation period to the decompensation period, various complications occur including coagulopathy, variceal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis (SBP), ascites, and hepatorenal syndrome (HRS). These complications pose increased mortality rates and are associated with a bad disease progression [3].

Signs and symptoms of decompensated cirrhosis include abdominal swelling, jaundice, gastrointestinal bleeding, fatigue, loss of appetite, nausea, peripheral edema, weight loss, pruritis, loss of libido, confusion, and drowsiness. A contracted, nodular liver, splenomegaly, ascites, dilated abdominal wall veins, spider angiomata, palmar erythema, peripheral edema, and asterixis are among the physical examination findings. Laboratory results may accidentally diagnose patients. Increased liver transaminase values, such as those for alanine and aspartate, are suggestive of persistent hepatocyte damage. Increased serum prothrombin time (PT, PTT) or International Normalized Ratio (INR) may signify that the liver is producing clotting factors less efficiently. Splenic sequestration may be indicated by thrombocytopenia. The level of total bilirubin may also be increased [4].

Portal hypertension is defined as portal pressure greater than 10 to 12 mm Hg. It is characterized by as an HVP G of more than 5 mm Hg in the hepatic veins with complications, such as ascites and varices, occurring at an HVPG greater than 10 mm Hg [5].

Varices are collateral veins that develop, in either esophagus or the stomach, as a result of the hepatic resistance of the blood that is coming from the splanchnic bed. Increased portal hypertension may result in the rupture of these varices. Consequently, bleeding from the upper GI occurs. About 20% of cirrhotic patients have gastric varices (GV) [6]. Upon discovery of "high-risk varices," primary prophylaxis with non-selective betablockers (NSBB) must be started. In secondary prophylaxis, the risk of rebleeding is lower with combination therapy for NSBBs + EBL than with monotherapy [7].

Treatment of acute variceal bleeding includes fluid resuscitation, vasoactive drug (octreotide) for three to five days after the bleeding stops, antibacterial prophylaxis with a third-generation cephalosporin (ceftriaxone 1 g/24 h), or a quinolone (norfloxacin 400 mg/12 h) for seven days. When endoscopy confirms acute variceal hemorrhage, variceal ligation should be carried out concurrently [7].

Ascites is a condition that causes abdominal distention due to an excess of fluid in the peritoneal cavity. Ascites is the most frequent cirrhosis consequence, appearing in 50% of patients within 10 years of diagnosis [8,9]. The most frequent reason for decompensation in cirrhosis is ascites. Renal sodium retention caused by the activation of sodium retaining systems (RAAS) and the sympathetic nervous system serves as the mainstay of ascites formation. When ascites is not infectious, resistant to treatment, or connected to HRS, it is not complex. Grade 1, Mild ascites: only an ultrasound examination can identify it. Grade 2, Modest Ascites: This condition causes a moderate amount of symmetrical abdominal distension. Grade 3: Significant abdominal distension is caused by large or massive ascites [7].

Patients who have experienced their first episode of grade 2 (moderate) ascites should only take anti-mineralocorticoid medication. beginning with 100 mg/day and gradually increasing (in 100 mg increments) every 72 hours. If lower doses are ineffective, the dose may be increased to 400 mg per day. Furosemide should be administered at an increasing stepwise dose from 40 mg/day to a maximum of 160 mg/day (in 40 mg steps) in patients who do not respond to antimineralocorticoids, as measured by a bodyweight loss of less than 2 kg/week, or in patients who develop hyperkalemia. When using diuretics, the most weight that can be lost is 0.5 kg/day in patients without edema and 1 kg/day in patients with edema is recommended. For patients with large ascites (grade 3), large-volume paracentesis (LVP) is the first line of treatment. Plasma volume expansion should be done after LVP to prevent post-paracentesis circulatory dysfunction (PPCD). To prevent PPCD in patients having LVP of more than 5 L of ascites, albumin (8 g/L of ascites removed) should be infused into the patient's bloodstream. Refractory ascites is advised to be treated first with repeated LVP plus albumin. Patients with persistent or recurrent ascites or those who are not helped by paracentesis should be assessed for transjugular portosystemic shunt (TIPS) implantation if they have loculated ascites [7].

A significant bacterial infection of the peritoneal cavity is called spontaneous bacterial peritonitis (SBP). The suspected mechanisms of translocation of the intraabdominal bacteria, Escherichia coli, Klebsiella pneumoniae, and Streptococcus pneumoniae, into the peritoneal cavity, are either they were transported via the lymphatic system or the blood circulation. SBP can be identified by doing a paracentesis and checking the fluid for a neutrophil cell count and bacterial culture. The neutrophil count ≥ 250 cells/mm3 and/or a positive bacterial culture are required for diagnosis. The initial treatment for SBP is broad-spectrum antibiotics empirically such as an intravenous third-generation cephalosporin until the antibiotic susceptibilities available. Based on a landmark trial reporting a significant reduction in mortality from 29% to 10% with albumin administration; albumin is suggested on days 1 (1.5 g/kg) and 3 (1 g/kg). To reduce the risk of recurrence and mortality following an episode of SBP, patients should be placed on secondary prophylaxis with alternatives such as ciprofloxacin, norfloxacin, and trimethoprim-sulfamethoxazole [10].

Hepatic Encephalopathy (HE) is a clinical term for brain dysfunction brought on by liver insufficiency, portal hypertension, and portosystemic shunting. Because of liver failure, detoxification of the toxic substances that are produced in the intestine is lost. Also, because of the portosystemic shunting, those substances enter the brain causing encephalopathy. It is classified into four grades based on brain function and the patient's symptoms. Lactulose is frequently used in the treatment and prevention of HE because it encourages the maintenance of bacteria that don't produce urease, which reduces the formation and absorption of colonic ammonia. The recommended daily goal for patients is three to four soft stools. Rifaximin, an antibiotic that is not absorbed and is used to reduce enteric bacterial flora, is a supplement drug for secondary prophylaxis. It has been demonstrated that using lactulose and rifaximin together reduces the number of hospitalizations [11].

Hepatorenal syndrome is a type of compromised kidney function that affects people with advanced liver disease as a result of the decreased kidney perfusion that results from this condition. According to established management guidelines, HRS should be treated with albumin, midodrine, and octreotide. The mortality rate has been significantly reduced by this regimen [10]. 20–40 g/day of albumin solution (20%) should be administered [7].

Patients with liver cirrhosis have a hemostatic imbalance with a high risk of both thrombosis and bleeding. Lack of coagulation factors, thrombocytopenia, platelet dysfunction, and a dysfunctional fibrinolytic system all increase the risk of bleeding. Factors II, VII, IX, and X, which depend on vitamin K, are most affected by coagulation factor deficiencies. Splenic sequestration and decreased production of thrombopoietin leads to thrombocytopenia. Risk should be evaluated on an individual basis because these processes differ from patient to patient [10]. All these factors contribute to the development of bleeding tendency and elevation of the INR of the patient. Vitamin K may be required to reduce the INR. It is not advised to use fresh frozen plasma to lower an elevated INR [11].

Case Presentation

We report a case of a 45-year-old man diagnosed with hepatic cirrhosis with hepatic encephalopathy grade 1. He came to the emergency department with watery diarrhea, severe fatigue, inability to perform minimal activities, decreased libido, anorexia, abdominal distention, discomfort and postprandial bloating, disturbance of sleep rhythm, and lower limb edema. He never consumed alcohol. The examination showed: 100/66 mmHg, blood pressure; 96 bpm, heart rate; 20 bpm, respiratory rate; 37C°, temperature; and bilateral lower limb edema. Abdominal ultrasound was performed and showed; shrunken cirrhotic liver, portal hypertension, moderate splenomegaly, and mild ascites. Laboratory work-up upon admission showed: normal AST and ALT, total bilirubin 2.5 mg/dL, direct bilirubin 1.04 mg/dL, albumin 23.2 g/L, potassium 2.5 mEq/L, and non-reactive HCV, HBsAg, and HIV. Hematological data on the day of admission were: Hb 8.1 g/dL, hematocrit 25.1%, red blood cell count 2.8*106/mm3, platelet 41*109/L, leukocytes 3.1*109/L, neutrophils 79%, lymphocytes 10%, monocytes 9%, eosinophils 2%, PT 23 seconds, PTT 62.3 seconds and INR 1.6.

As seen in the lab results, the patient's liver problems have resulted in; hypoalbuminemia, coagulopathy, anemia, thrombocytopenia, and leukopenia (pancytopenia). Hypokalemia is assumed to be due to diarrhea. Also, the patient's symptoms clearly indicate hepatic encephalopathy grade 1 and moderate ascites.

Calculating Child-Pugh score for this patient: total bilirubin = 2.5 mg/dl (2 points), serum albumin = 2.3 g/dl (3 points), prolonged prothrombin time = 7 sec. (3 points), ascites: mild (2 points), hepatic encephalopathy: grade 1 (2 points). Thus, this patient has a total Child-Pugh score of 12 which refers to class C. Therefore, he has a one-year survival of 45% and two-year survival of 35%.

On the day of admission, the patient was given; dextrose 5% three times daily, as nutrition and hydration; cefotaxime 1g three times daily and moxifloxacin 400 mg infused one time daily, as prophylaxis for spontaneous bacterial peritonitis; rifaximin 550 mg twice daily orally, and metronidazole 500 mg infused three times daily, as a treatment for hepatic encephalopathy; vitamin K 5mg injected subcutaneously two times daily, as coagulopathy management; potassium chloride 40 mEq infused in normal saline three times daily, for the hypokalemia; ornithine 250 mg three times daily, to enhance ammonia elimination; and selenium once daily orally, to prevent further liver necrosis.

On day 3 after admission, the patient was given octreotide 50 mcg infused twice daily for seven days and it was held on day 10 after admission. On day 10 after admission, his sodium level was 124 mEq/L, so he was given 500 mL of normal saline 0.9% for two subsequent days. Also, lactulose 30 mL three times daily orally was added to his regimen.

The patient's blood pressure reading on day 11 following admission was 150/90. At 9:45 AM, blood work was performed and showed: Hb 7.9 g/dL, leukocytes 2.6 *109 /L, RBC count 2.9*106 /mm3, and platelet 53 *109 /L. On the same day, the patient bled from his upper GI tract and at 4:58 PM his blood work showed a drop in Hb to 7.3 g/dL, leukocytes to 1.78 *109 /L, and RBC to 2.75*106 /mm3. This indicates bleeding. He was given carvedilol 3.125 mg twice daily. His cefotaxime and moxifloxacin were held and substituted with imipenem 1 gram three times daily. Furthermore, acetylcysteine 300 mg orally was added to his regimen.

On day 12 after admission, his serum albumin levels were 15 g/L and his potassium levels were 2.7 mEq/L. He was given albumin to correct his hypoalbuminemia. He also received one unit of packed RBCs on day 13 following hospitalization. On day 16 after admission, his serum albumin level was found to be 23 g/L owing to albumin administration.

On day 17 after admission, at 9:20 AM, his Hb was 9.1 g/dL. And at 1:28 PM it dropped to 8.8 g/dL due to upper GI bleeding. No drug was discontinued, added, or modified.

Discussion

Mild ascites is only detected by ultrasound, moderate ascites, however, is manifested by moderate distension of the abdomen [7]. In this patient, although ultrasound showed mild ascites, abdominal distension and fullness flank indicate moderate ascites.

Despite the moderate ascites, peripheral edema increased blood pressure, and hypokalemia, which may be improved by potassium-sparing diuretic, this patient was not prescribed any diuretic. This patient is a good candidate for receiving spironolactone owing to his hypokalemia which may be improved by spironolactone. However, if there is no improvement with lower dosages, patients with moderate ascites should get an anti-mineralocorticoid medication alone starting at 100 mg/day and increasing stepwise every 72 hours (in 100 mg steps) to a maximum of 400 mg/day[7].

When "high-risk varices" are found, the primary prophylaxis against variceal bleeding must begin (i.e. Child-Pugh C patients) due to the increased risk of variceal hemorrhage. According to American Association for the Study of Liver Diseases (AASLD) recommendations, individuals with cirrhosis who have high-risk varices should take non-selective beta-blockers for primary prophylaxis of variceal bleeding, along with band ligation for secondary prophylaxis [7].

Although this patient has a history of upper GI bleeding and is at high risk for variceal bleeding (child-pugh C), he had not been on any prophylactic beta-blockers, nor was he prescribed one on the day of admission. Instead, carvedilol was prescribed on day 11 after admission, when he bled from his upper GI. Not to mention that carvedilol is not the first choice beta-blocker for patients with decompensated cirrhosis because it is suspected to cause a greater drop in the mean arterial pressure. Propranolol or nadolol, however, is the first choice for patients with decompensated cirrhosis [12].

Moreover, in patients with an intercurrent acute condition such as bleeding, AKI, or SBP, beta-blockers should be discontinued and after recovery, they can be reinitiated [7]. In this case, however, carvedilol was initiated on day 11 after admission. The patient bled on day 17 but carvedilol was never held.

As the initial course of treatment for acute variceal bleeding, the use of vasoactive medications in conjunction with ligation is advised [7]. This patient was given octreotide for 7 days (from day 3 to 10 after admission) and held. The day after octreotide was held (day 11) the patient bled. Octreotide was not reinitiated nor was band ligation performed.

The first line of treatment for episodic hepatic encephalopathy is lactulose. For the prevention of recurrence of hepatic encephalopathy, rifaximin is an effective adjunct therapy to lactulose. However, metronidazole is an additional option for the treatment of hepatic encephalopathy [13]. On the contrary, this patient was first given rifaximin and metronidazole on the day of admission, and three days later, lactulose -which is the first choice was added to his regimen. The three medications were continued despite the no need for metronidazole – the alternative choice.

Although it has a hepatoprotective effect, acetylcysteine use is limited to acetaminophen overdose or acetaminophen-related acute liver injury. In this case, however, the patient was given acetylcysteine despite the fact that his diagnosis was not acetaminophen-related nor was it acute, not to mention the cost-effectiveness of the patient's drug regimen.

Conclusion

Medical negligence refers to action by medical professionals that violates their obligations to patients by failing to provide

adequate care or take appropriate precautions. It happens as a result of the patients' being treated negligently, carelessly, or incorrectly. This case illustrated that some healthcare providers don't give the expected standard therapy. Therefore, we believe it is essential to update the healthcare system, improve hospital working conditions, and create and follow clinical practice guidelines. We believe that a focus on the application of diagnosis and therapy protocols, standards, post-graduation education, clear patient education, and improved patient communication is absolutely essential.

Conflict of Interest: The authors have no conflicts of interest to declare.

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