Treatment of cirrhosis with hepatic encephalopathy nursing essay

J. L is a 61 year old male who arrived at the emergency department with his wife. J. L is confused and having difficulty doing simple tasks since yesterday. He is alert and orientated times three but is displaying apraxia and associated agnosia. His wife stated that he was having trouble using the bathroom because he couldn't remember what it was for and he couldn't carry out purposeful movements. His wife stated he has no other deficits except that his speech is slower today. His vital signs on admission are 114/64 – 56 – 97. 7F – 20 – 97%RA. Bilateral crackles were heard in lung fields, increased respiratory effort is noted but no more than the patient's baseline. His chest x-ray only showed pulmonary fibrosis. His abdomen was mildly distended and he complained of some tenderness. His skin was dry, warm and pink and there is no edema noted. On admission his BNP, bilirubin, and ammonia were elevated.

Primary diagnosis/priority secondary diagnosis

The primary diagnosis is cirrhosis with hepatic encephalopathy and his secondary diagnosis is Congestive Heart Failure (CHF).

History

He has a medical history of pulmonary fibrosis, hepatic cirrhosis, hypothyroidism, splenomegaly, diabetes mellitus type two, CHF, hypertension (HTN), and pancytopenia.

Pathophysiology of the primary diagnosis

Cirrhosis is scaring on the liver or inflammation of the liver. Inflammation results in destruction of hepatocytes. Fibrotic bands of connective tissue and nodules develop throughout the liver that can block bile ducts and blood

flow. At first the liver is enlarged and hard but as the damage progresses the liver gets smaller (Ignatavisius & Workman, 2006). The cirrhosis caused elevated serum ammonia and because of this the patient developed hepatic encephalopathy. Elevated ammonia is the result of the shunting of portal venous blood into the central circulation so that the liver is bypassed. Toxic substances are normally broken down in the intestines. When the liver cannot convert ammonia to urea, the ammonia crosses the blood brain barrier (Ignatavicius & Workman, 2006).

Pathophysiology of the secondary diagnosis

Congestive heart failure occurs when the heart does not pump well enough to supply the body with blood. As a result blood starts to back up into the body. With right sided failure the blood backs up into the peripheral extremities and can cause edema. For left sided failure the blood backs up into the lungs and causes pulmonary edema. CHF can result from any functual or structural cardiac disorder that impairs the ventricles ability to fill with or eject blood. The two mechanisms of reduced cardiac output are systolic dysfunction and diastolic dysfunction (Figueroa & Peters, 2006).

Etiology and clinical manifestations

The most common causes of cirrhosis are an increase in alcohol consumption, morbid obesity and chronic viral hepatitis (Sargent & Martin, 2006). Other causes are autoimmune hepatitis, streatohepatitis, drugs and toxins, bilary disease, metabolic/genetic causes, and severe right sided heart failure (Ignatavicius & Workman, 2006). Cirrhosis or liver failure can lead to hepatic encephalopathy. Factors that may precipitate hepatic encephalopathy are infections, drugs such as hypnotics, opioids, sedatives,

analgesics and diuretics, GI bleeding because it causes a large protein load in the intestines, constipation, hypokalemia, hypovolemia and a high protein diet (Ignatavicius & Workman, 2006). There are four stages of hepatic encephalopathy which are prodromal, impending, stuporous and comatose. Prodromal signs and symptoms are personality changes, behavior changes, inability to concentrate, fatigue, drowsiness, and slurred slow speech. Impending signs and symptoms are continuing mental changes, mental confusion, disorientation, and asterixis. Stuporous signs and symptoms are progressing mental confusion, stuporous, abnormal EEG, muscle twitching, and hyperreflexia. Comatose signs and symptoms include unresponsiveness, positive babinski's sign, muscle rigidity and seizures (Ignatavicius & Workman, 2006, p. 1370).

There are many causes of CHF. Some are damaged heart valves, blocked blood vessels, toxic exposures, infections, hypertension, pericardial disease, congenital heart disease, prolonged arrhythmias, or unhealthy lifestyle habits. Some unhealthy lifestyle habits that may contribute to CHF are smoking, drinking excessive alcohol, obesity, lack of physical activity and years of uncontrolled high blood pressure. Early symptoms are shortness of breath, cough, or not being able to get a deep breath. Other symptoms are exercise intolerance, fluid retention, edema in the lower extremities and fatigue. A person with CHF may awaken at night unable to catch their breath. A frothy pink liquid may be coughed up as the buildup of fluid in the lungs becomes more severe (Ignatavicius & Workman, 2006).

Relationship

CHF can ultimately lead to damage of other organs. The heart is not able to pump effectively. This could lead to hypoxia. The liver may not have been getting enough oxygenated blood which would lead to a damaged liver. If the liver is damaged and not working properly, it cannot convert ammonia to urea causing elevated levels of ammonia in the blood which crosses the blood brain barrier causing neurologic signs and symptoms. The patient's ammonia level was 69(n= 9-33). This is very high and is why the patient was confused and unable to do simple tasks. The patient also had slowed speech as a result of this.

Actual or potential impact of relevant medical/surgical history on the primary diagnosis and priority secondary diagnosis

Pulmonary fibrosis reduces the amount of oxygen in the bloodstream causing hypoxemia which will eventually lead to hypoxia. Chronic hypoxia will damage the organs such as the liver. Pulmonary fibrosis can also cause right sided heart failure. This may have caused or worsened the patients CHF. (mayoclinic, pulmonary fibrosis, 2009). Because the patient had pulmonary fibrosis and CHF there would be further damage to the body due to the lack of oxygen.

Hypothyroidism could connect to cirrhosis because the thyroid hormones regulate the metabolic rate of hepatocytes. Inadequate thyroid function will eventually damage the liver (Malik & Hodgson, 2006). Also, the patient's heart is not working properly so there is a decrease in oxygenated blood.

There would be more damage to the organs without proper oxygen and without proper thyroid hormones.

The patient’s splenomegaly is the cause of the pancytopenia. The enlarged spleen destroys platelets, causing thrombocytopenia. Cirrhosis can cause an enlargement of the spleen because blood is backing up into the spleen (Ignatavicius & Workman, 2006). With a CHF patient we want to reduce the workload of the heart. If the spleen is enlarged and the red blood cells are reduced the heart will want to work harder to make up for this. That will increase the workload on the heart and cause more damage.

Patients with diabetes are at a greater risk for developing heart disease (Ignatavicius & Workman, 2006). The liver plays a role in regulating carbohydrate metabolism. If the liver is damaged, blood glucose cannot be maintained (Levinthal & Tavill, 1999). This may be why the patient’s glucose levels were so high.

Hypertension is common in people with diabetes. Having high blood pressure and diabetes increases the risk for heart disease. The heart has to work harder to pump blood against the increased pressure in the arteries. This can cause the heart muscle to thicken or the left ventricle to weaken causing heart failure. Because of the increased pressure in the arteries, the heart may not adequately get oxygenated blood to the organs. The liver is already damaged so hypertension will increase this damage.

Combined effect

Pulmonary fibrosis, hypertension and diabetes probably lead to the patient's CHF. The CHF played a role in the liver damage because of hypoxia. The liver damage could have caused the diabetes, splenomegaly and pancytopenia.

Medical Management

Recommended textbook interventions

The goal for cirrhosis with hepatic encephalopathy is to lower the serum ammonia level. Serum enzymes such as AST, ALT, LDH, ammonia and bilirubin would be elevated. Serum proteins such as albumin and globulin will be abnormal. PT or INR will be prolonged (Ignatavicius & Workman, 2006). X-rays, CT scan, and an ultrasound will be ordered to determine enlargement of the liver or spleen and ascites. A MRI will determine if there are masses on the liver and a biopsy of the liver may be required to determine the extent of the disease. If there are esophageal varices suspected an EGD would be ordered. An EEG may be ordered for patients with hepatic encephalopathy. Collaboration with the dietician is needed for diet therapy. The diet for a patient with cirrhosis consists of high calorie, high carb, low sodium, low to moderate fat and high protein. The diet for a patient with elevated ammonia levels usually includes low protein foods and simple carbs. Vegetable protein may be more beneficial than animal protein. Increased amounts of ammonia can cause GI bleeding. In this case the patient would have to be NPO and using a nasogastric tube (Ignatavicius & Workman, 2006). Activities for the patient would be based on the symptoms the patient has. If there is an increased fluid accumulation the patient may have respiratory problems in which case the patient should sit up to facilitate breathing. A patient with

hepatic encephalopathy should ambulate only with someone present due to confusion and weakness.

Actual interventions and patient response

The patient’s labs were drawn. The bilirubin was 2. 7(n= 0. 2-1. 3) showing that the liver cells are damaged. Serum ammonia was 69(n= 9-33) which explains the hepatic encephalopathy. Because of the heart failure the BNP is 260(n= 0-99). The clotting factors came back abnormal because of hepatic cell damage. PT was 14. 7(n= 9-12), INR was 1. 4(n= 1), and platelets were 45(n= 150-450). The patient had a chest x-ray that only showed diffuse pulmonary fibrosis. The head CT showed no intracranial process and the EKG showed sinus bradycardia with T wave inversion and ST segment depression. The renal ultrasound and pelvic CT were negative. The patient was put on a 2000 ADA diet requiring supplemental carnation instant breakfast to increase calories. In regards to activity the patient was encouraged to ambulate as tolerated with one assist. He walked around his room when someone was there with him due to the weakness and confusion.

Nursing Management

Recommended textbook interventions

It is important to obtain a medical history from the patient. A complete nursing assessment should be done every shift paying especially close attention to worsening signs of hepatic encephalopathy such as irritability, restlessness, lethargy and change in behavior. Signs such as a distended abdomen, yellow skin, and itchy skin would indicate worsening cirrhosis. Vital signs should be taken every four hours and input and output should be

monitored due to the risk of fluid overload. Monitoring the telemetry, and labs such as ammonia, liver function and electrolytes are also important. The nurse should teach the patient the purpose of each medication, the consequences of not taking the medications, and the side effects such as diarrhea due to the Lactulose. The patient should be taught to take the prescribed medications and not to stop taking any abruptly. The Lactulose syrup should cause three to five bowel movements every day. If experiencing weakness, irregular heartbeat, or light-headedness, the patient should call the health care provider right away. The nurse should also teach the patient and his wife about safety due to the weakness caused by the elevated ammonia. The patient is taught to use the call light before getting out of bed to reduce the risk of falls. Diet therapy should be addressed and the patient should be taught to eat a low sodium diet, eat small frequent meals, and follow the 2000 ADA diet with added instant carnation (Ignatavicius & Workman, 2006).

Actual interventions and patient response

The assessment initially revealed crackles in the bases and shortness of breath. The abdomen was a little bit distended and tender, and the skin was pink, warm and dry. The input and output continued to be equal. The patient tolerated small portions of meals and substituted them with carnation instant breakfast. While at the hospital the patient’s ammonia dropped from 69 to 39(n= 9-33). The patient’s level of consciousness also improved. Upon arrival to the hospital the patient was confused and lethargic. The patient also talked very slowly. It took two people to transfer the patient to bed. On day three the patient was no longer confused and talked normally. The

patient was also able to get out of bed and walk around. He was able to perform tasks without difficulty such as using the bathroom and brushing his teeth. Initially the patient did not participate in the teaching but the wife was at the bedside and listened carefully and asked questions and expressed understanding.

Pharmacological Management

Recommended textbook interventions

Drug therapy is more difficult because the liver cannot metabolize drugs as well because it is damaged. Analgesics, sedatives and barbiturates should not be used. There are three types of drugs that can lower ammonia. They are Lactulose, Neomycin sulfate, and Metronidazole. Lactulose is a sweet liquid that causes a laxative effect. The goal is to rid the ammonia in the stool. It also decreases the ph in the bowel causing the ammonia to leave the circulatory system. The patient should have three to four non- watery bowl movements per day. Neomycin Sulfate is a broad spectrum antibiotic that destroys the normal flora in the bowel diminishing protein breakdown and decreasing the rate of ammonia production. Metronidazole is a broad spectrum antibiotic with similar action as neomycin but has less potential for renal toxicity (Ignatavicius & Workman, 2006).

Actual interventions and patient response

The patient was put on Lactulose 15 mg bid until diarrhea. He had many stools but did not have diarrhea. Lactulose was continued throughout the hospital stay and he was discharged on 30mg Lactulose once a day. A onetime dose of Lasix (diuretic) was given to decrease the crackles in the

lungs. After the Lasix the lung sounds were clear. Initially, the patient’s blood pressure was low, causing the Metoprolol (beta-blocker) to be held for one day. This improved the blood pressure before discharge.

Nursing care plan

P Alteration in thought process

R Increased serum ammonia

C Ammonia 69, lethargy, inability to perform simple tasks such as using the bathroom

Goal

The patient will have an improvement in thought process as evidenced by being able to perform simple tasks such as using the bathroom, not being lethargic and serum ammonia being within the normal limits of 9-33 at the time of discharge.

Three nursing interventions

The nurse will monitor vital signs and level of consciousness every four hours, paying close attention to signs of worsening hepatic encephalopathy including restlessness, irritability and inappropriate behaviors.

The nurse will administer Lactulose to decrease intestinal ammonia levels and monitor ammonia levels.

The nurse will teach patient that the Lactulose will cause three to five bowel movements per day and to let the nurse know if they are watery.

Evaluation

The goal was partially met on the day of discharge. Because the ammonia level dropped to 39(n= 9-33), the patient will continue on the Lactulose at home. The patient was no longer lethargic and was able to walk around by himself. He could also perform tasks such as using the bathroom.

Provider and manager role

Four tasks that I implemented in my care of this patient specific to the provider of care role are; collected and utilized relevant data such as labs, diagnostics and pertinent medical history, correlated changes in status, called the doctor when the patients ammonia came back high, developed and implemented the teaching plans based on the clients strengths and barriers, and implemented physician orders with the supervision of the preceptor.

Members who participated in the patients care were the physician, discharge planner, social worker, secretary, phlebotomist, dietician, RN and LPN. The physician was responsible for prescribing medications to lower the ammonia, keep the blood sugar normal, and keep the blood pressure normal. The phlebotomist drew the patient’s blood when the labs were due. The dietician was to make sure the patient’s diet could meet his nutritional needs. The RN had to assess the patient, implement the plan of care, establish goals and evaluate the goals. The LPN assisted the RN with tasks such as bathing, vitals, daily weights and recording input and output. The discharge planner met with the patient and his wife to make sure their needs were going to be met after they were discharged. The patient was discharged home with everything he needed. The secretary kept the chart up to date and made

sure the labs and diagnostic tests were ordered. The social worker met with the patient and his wife and talked through any concerns they may have had.

As manager of care, I had to establish priorities of patient care based on how he was doing. The manager of care is also responsible for delegation so the vital signs were delegated to the LPN and I made sure to follow up to make sure they were within normal limits. As manager of care, I also interacted with other members of the health team and acted as a patient advocate when needed.

This experience really contributed to my growth as manager of care. I am used to doing assessments and getting vitals without much thought in my previous job as a nurse’s aide. In this role at the hospital I have to think about what it all means relative to my patient’s status. I had to not only look at the ammonia levels but think about what they mean to my patient and what signs and symptoms my patient has. I really had to bring everything together to look at the patient as a whole. I had to address medications, labs, and other medical diagnosis. I had to think about what everything as a whole meant for the patient. I also learned about the importance of teamwork. The doctor wouldn’t know the ammonia was high without the lab to check the blood and the nurse to call the doctor with the results. Dietary made sure the patient was getting adequate calories. Communication is very important to give the best care. I learned that as a nurse in the manager role, I have to make sure all parts of the team are well informed of the patient’s status and needs.