RMIT University

School of Health Sciences: Discipline of Nursing

NURS2096 Acute Care Nursing 2 2015

Liver Disease Case Study Assessment

History of Present Illness

Mr McGrath is a 46-year-old university lecturer with a 25-year history of heavy drinking; 5 years ago he was diagnosed with alcoholic cirrhosis of the liver. He stopped drinking after his diagnosis.

Mr. McGrath went to his GP with his wife complaining of fatigue, weakness, and pain under his right rib cage. He had been complaining to his wife for a couple of months but has refused to see anyone about it until now. He has been self-medicating with panadol for the pain. According to Mr. McGrath's wife he has been more difficult than usual for the past week "very confused and acting strangely". His wife was concerned this morning as he didn't seem to understand her. His wife also thinks his stomach is swelling up again and is worried his cirrhosis is getting worse.

He has recently gained 10kg, and his abdomen has become significantly swollen. Mr. McGrath has had difficulty sleeping, remembering things, and has according to his wife been more grumpy than usual. Over the week, Mr. McGrath has become increasingly lethargic and disoriented. Based on his presentation, Mr. G was admitted to the hospital with abdominal swelling and confusion for investigation.

Past Medical/Surgical History

Regular bouts of Pneumonia over the last 6 years, alcoholic cirrhosis 5 years ago, anemia, an admission for upper GI hemorrhage secondary to oesophageal varices 3 years ago. Admitted for abdominal paracentesis 18 months ago. He has had a laparoscopic cholcystectomy 15 years ago. Appendectomy at 16, fractured arm at 13years old.

Family History

Mother died of liver disease at age 64, father died of a heart attack at age 58.

Social History

MrMcGrath has been married for 20 years with 3 daughters and a son. Previously a heavy drinker (6 slabs of VB /week x 20 years) stopped drinking 5 ago. Smokes 1 pack per day has been smoking for 30 years.

Medications

- Propranolol 10 mg orally 8/24
- Spironolactone 50 mg orally BD
- Furosemide 20 mg orally BD
- OTC drugs – Panadol – for his pain not prescribed

On examination

Mr. McGrath is restless and disoriented to person, place, and time. He responds to questions slowly, and his answers are often inappropriate.

Mr. McGrath's skin and sclera have a yellowish colour, and he has several ecchymoses to the lower extremities. PERRL. Lungs clear to auscultation. Abdomen distended, firm, and tender with prominent veins at the umbilicus. Bowel sounds normal. Enlarged liver. Haemorrhoids present. Slight metabolic flap observed. Confused and disoriented.

BP 118/70, P 82 and regular, RR 22, T 37.7°C, weight 95Kg, height 185, SaO2 91% (room air)

Biochemistry:

Serum albumin 25g/l	AST 120 U/L
Total bilirubin 55umol/l	ALT 80 U/L
Prothrombin time 20 seconds	ALP 170 U/L
GGT 110 U/L	

Review the presentation and management of Mr McGrath ensuring you address the following questions:

- **Explain the cause of his presenting symptoms particularly in relation to his confusion.**

  McGrath presented with recent weight gain, swollen abdomen, difficulty in sleeping, difficulty in remembering things and behavioral symptoms like irritability, sulkiness,

lethargy and disorientation. He presented with abdominal swelling and confusion for investigation as well. This may be due to the disease liver encephalopathy. Hepatic or Liver encephalopathy is the deterioration of the function of brain and subsequent nervous system damage due to the toxic buildup of certain substances in the blood, which are normally removed by the liver. Symptoms may include lowered consciousness, disorientation, and changes in logical thinking including other behavioral changes. Liver failure leads to this state of and in the serious advanced stages it may lead to hepatic coma also called coma hepaticum. The condition is very serious and may ne life threatening. The diagnosis can be detected by neuropsychological testing as well as it is difficult to be tested by laboratory testing. The first stage of hepatic encephalopathy is observed with an inverted sleep-wake pattern. In the next stage ie, second, lethargy and sluggishness appears in the patient. The third stage comes with worsened state of confusion and the fourth stage is marked by coma.

- **Describe the pathophysiology of the development of oesophageal varices and discuss potential management strategies for bleeding varices.**

  Esophageal varices are abnormally enlarged veins in the lower portion of the esophagus. Esophageal varices are seen most commonly in people suffering with liver diseases. These varices are seen to develop as a result of obstruction to the blood flow by scar tissue in the liver or obstruction by a clot. In order to escape and find a way out of the blockages, there is a blood flow into the smaller surrounding blood vessels around the blockage that are not intended for carrying large blood volumes. Due to this the vessels may be seen to leak blood or rupture, resulting in serious bleeding which may be a life-threatening condition. Variceal bleeding is a major complication of portal hypertension and also variceal bleeding is the leading cause of death in patients with cirrhosis (Graham & Smith, 1981; Gines et al., 1987; El-Serag & Everhart 2000; McCormick & O'Keefe 2001). Around 70% of all upper-gastrointestinal hemorrhages in liver cirrhosis are caused by this rupture of esophageal varices (D'Amico & De Franchis, 2003). Hence we can say that a variceal origin can be considered if any any cirrhosis patient presents with symptoms of gartrointetsinal bleeding (that too before endoscopy is done). Many randomised controlled trials report that about 40 to 50 % of these hemorrhages spontaneously stop bleeding (D'Amico et al. 1999). Current available therapeutic choices are known to control the variceal bleeding in up to eighty percent of the total patients. Early mortality after acute variceal bleeding episode is as high as up to 25 %.

Bleeding varices can be managed using various therapeutic approaches. Developments in this field have resulted in significantly improved diagnosis and prognosis over last twenty years. A number of drugs and medical procedures can help prevent and stop bleeding from esophageal varices. The goal of treatment of bleeding varices is to stop the bleeding quickly. Bleeding must be controlled quickly to prevent shock and death.

In case of excessive bleeding the patient may be put on a ventilator in order to prevent the going down of blood into the lungs and to protect the airways.

To treat acute bleeding:

- A clotting agent may be injected in the veins and even some band may be placed around the veins done by using endoscope.
- Any vasoconstricting agent may also be used to stop the bleeding. These agents are known to make the blood vessels tight. Some vasoconstricting agents are octreotide, vasopressin etc.
- Sometimes we may insert a tube through nose into the stomach and inflate using air. This causes pressure inside the vessels and may cause more stress towards the bleeding veins.
- As soon as the bleeding stops, the varices may now be treated using medications and other protocols to further prevent such bleeding.
- The medicines used include certain beta blockers, like propranolol and nadolol that are known to lower the bleeding risk in future.
- One of the medical procedures used for treatment include transjugular intrahepatic portosystemic shunt (TIPS) which is the procedure for creating new connections between the blood vessels in liver. This procedure is known to lower the present in veins and thus lower the risk of future bleeding.
- In case other therapeutic modalities fail, the heathcare providers may then take help from emergency surgeon. The emergency surgery options include portocaval shunts or removal of the esophagus.
- Patients with bleeding varices (caused by the liver disease) may even need a liver transplant.

- **Discuss the role and potential complications of abdominal paracentesis in the management of ascites.**

Paracentesis is the procedure in which a needle /catheter is inserted into the peritoneal cavity and then the ascitic fluid is extracted in order to help in diagnosis and therapy. Ascitic fluid is used for helping in determining the etiology of ascites, and also subsequently for evaluation of some infection. The fluid can also be used to determine the presence of any form of cancer in the patient. Ascites (the fluid filled) may be caused by some form of infection, inflammatory insult or some injury, or even cancer or the liver cirrhosis. The procedure involves taking the fluid out of the abdomen using a long needle/catheter. The abdominal paracentesis may also be done in cases to take out the fluid and relieve the abdominal pressure or even pain in the patient (in liver cirrhosis or cancer).

The commonest site for taking out the ascitic fluid using the paracentesis procedure tap is about15 cm lateral of the umbilicus. While doing this, care has to be taken to avoid enlarged liver/spleen. Complications of the abdominal paracentesis are seen to occur in less than 1% of patients. These complications are abdominal haematomas and they are rarely life threatening. More severe complications like haemoperitoneum or bowel perforation are extremely rare (around 1/1000). Ascitic fluid leak is one of the commonest reported complications of abdominal paracentesis. Such ascetic leaks occur in case when a Z-track (while performing the procedure) has been done incorrectly or when a large-bore needle is used for the procedure. Sometimes bleeding may occur as a result of wrongly performed abdominal paracentesis. An acute abdomen in need of surgery is an absolute contraindication to sbdominal paracentesis. Severe thrombocytopenia (platelet count <20 × 103/μL) and coagulopathy (international normalized ratio [INR] >2.0) are relative contraindications.

- **Discuss the educational requirements for Mr McGrath following his current admission.**

  Mr. McGrath needs to be counseled and educated by the heathcare providers as the role of counseling is integral to such cases. He has history alcoholic cirrhosis, anemia along with upper GI hemorrhage secondary to oesophageal varices and, appendectomy. These complications and their therapeutic follow up needs proper education of the patient by the heathcare providers. Also in the present case educating the patient will help him understand the seriousness of his life threatening condition. The case looks like proceeding to Hepatic encephalopathy which is known to cause hepatic coma. This can even lead to death. So, keeping in mind the seriousness of the disease we need to educate the patient accordingly.

- **Analyze the drug regimen Mr McGrath is currently prescribed and identify the rational / reason he has been prescribed these drugs.**

  The medications prescribed to him include Propranolol (10 mg orally 8/24), Spironolactone 50 mg orally, Furosemide 20 mg orally, OTC drugs – Panadol – (for his pain not prescribed). Propranolol is known to decrease the portal pressure and also inhibits the secretion of renin in patients with chronic liver disease, this subsequently lowers the tendency to ascites formation. But it is also seen that beta-blockers like Propranolol may have a serious impact on the survivorship of patients with cirrhosis and refractory ascites. Another medicine prescribed was spironolactone which is a diuretic. Diuretics which act by blocking the aldosterone receptors are usually preferred medicine because of the presence of hyperaldosteronism in patients with cirrhosis. Loop diuretics can be used in combination, but these are seen to be ineffective if used alone. The initial starting dose of spironolactone is usually 100 mg once daily and can be titrated up to a maximum of 400 mg once a day. Absorption of spironolactone is improved if administered with food. Spironolactone is known to be a weak diuretic and thus require combination of one loop diuretic such as furosemide. Its effects are seen within 30 minutes of administration orally. It is an effective diuretic but is not as effective as spironolactone alone.

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