

End Stage Liver Disease in the ICU: Walking a Tightrope

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LEARNING OUTCOMES

After completing this chapter you will be able to:

1. Discuss the pathophysiology of liver failure
2. Describe clinical manifestations of liver failure
3. Describe nursing implications, diagnostics and treatment required in the patient experiencing liver failure.

INTRODUCTION

Patients who suffer from end stage liver disease (ESLD) can present many challenges to the nurses caring for them. While they may appear to be stable, they are balancing on a very fine tightrope and life threatening complications can arise suddenly, requiring immediate attention to prevent catastrophic outcomes. The critical care nurse must be able to thoroughly assess for impending complications and always be prepared for a change in the patient's condition.

The liver plays many roles in maintaining health. It clears the body of ingested toxins, manufactures numerous clotting factors, helps to maintain vascular oncotic pressure by producing albumin and plays a role in glycemic control. As the liver fails, it can no longer carry out these normal functions leading to the complications that are associated with ESLD.

While the liver has a remarkable ability to recover and regenerate, when severe injury occurs, whether from disease, accumulation of toxins, such as alcohol, infection, such as chronic hepatitis, or other disorder, this regeneration can lead to fibrotic changes in the liver tissue. Once enough of the liver tissue becomes diseased and irreversible changes have occurred, a diagnosis of cirrhosis is made. These cirrhotic changes lead to an inability for normal blood flow through the liver as well as an inability for the liver to carry out its normal functions. The point is reached when there are not enough healthy hepatocytes remaining. The complications that are seen in ESLD are a direct result of these structural changes.

HEPATIC ENCEPHALOPATHY

Hepatic encephalopathy (HE) occurs in patients with ESLD as a result of a build-up of toxins able to cross the blood brain barrier and leads to changes in mental status. The exact cause of HE is unclear but there is strong association between elevation of circulating ammonia and severity of HE. Intestinal bacteria breakdown protein in the intestine leading to an increase in ammonia. When the cirrhotic liver is unable to metabolize the absorbed ammonia, it enters the systemic circulation and is able to cross the blood brain barrier, leading to changes in neurotransmitters and alteration in neurological function (Scott, 2014).

Hepatic encephalopathy is categorized by type and severity. There are 3 types of HE, type A which is associated with acute liver failure, type B associated with abnormal ammonia metabolism and no underlying liver disease, and type C associated with chronic liver failure (Leise et al., 2014).

Hepatic encephalopathy is further defined by grades (Table 1). Once a patient deteriorates to Grade 2, transfer to the ICU is warranted, as a continuous decline can lead to inability to protect the airway and respiratory failure. These patients require intubation and mechanical ventilation until their mental status improves.

Worsening HE can occur in patients who develop gastrointestinal (GI) bleeding or an infection. These patients must be closely monitored for decline in their mental status and the need for airway management (Kelso, 2008). Lactulose can be used orally or via enema to trap ammonia and expel it from the GI tract. Doses should be titrated to three to four loose stools per day (Leise, 2014). Rifaximin, an antimicrobial, can be used to decrease GI bacteria which can decrease the amount of ammonia that is produced from digestion of protein. The usual dose is 550 mg two times per day (Scott, 2014).

Index	Clinical signs and symptoms
1	Mild changes in personality, decreased attention span, loss of calculating functions. No asterix present
2	Worsening personality changes, disoriented, incontinence, asterix can be seen, reversed sleep/wake patterns
3	Somnolent but arousable from sleep, obvious asterix, very confused with slurred, incoherent speech
4	Comatose and may or may not respond to noxious stimulation, may see posturing

Table 1. Grades of hepatic encephalopathy

PORTAL HYPERTENSION

As cirrhotic changes in the liver worsen, blood can no longer flow freely through the liver lobules. There is an increase in venous pressure leading to increased pressures within the portal system. The development of portal hypertension contributes to a number of the complications we see in patients with ESLD including, ascites, gastroesophageal varices and variceal bleeding.

Ascites

Ascites occurs when protein rich fluid accumulates in the peritoneal cavity. The increased pressure in the portal system increases the hydrostatic pressure in the vessels causing fluid rich in protein to be forced out of the vascular space into the abdomen. This increases the oncotic, or water-pulling, pressure within the abdomen causing further fluid to be pulled in (Kelso, 2013).

Complications associated with ascites include infection, notably spontaneous bacterial peritonitis (SBP), and respiratory failure, occurring when enough fluid accumulates within the abdominal cavity to impede adequate respiratory function. SBP should be suspected in all ESLD patients who have any amount of ascites and develop a fever and abdominal pain (Kelso, 2008). A diagnostic paracentesis can be performed to look for white blood cells and bacteria in the ascites. If an infection is present, antimicrobials need to be started and the ascites may need to be completely drained from the peritoneal cavity.

Typically, ascites is controlled with diuretics. If enough fluid accumulates to compromise respiratory function, a therapeutic paracentesis may be performed to drain enough ascites to prevent the need for intubation and mechanical ventilation.

Gastroesophageal varices

Although varices can occur anywhere within the splanchnic circulation,



these collateral vessels are typically seen in the distal esophagus and the gastric fundus (Garcia-Tsao et al., 2007). An esophagogastroduodenoscopy (EGD) is used not only to diagnose varices, but to determine their severity and the likelihood of bleeding. Banding of problematic vessels can occur in order to prevent, or decrease the risk of a variceal hemorrhage (Garcia-Tsao et al., 2007). Beta blockers may also be used to decrease portal pressure and decrease the risk of bleeding, however patients must be closely monitored for systemic hypotension that may complicate this treatment option (Garcia-Tsao et al., 2007).

Variceal hemorrhage

The rupture and bleeding from varices is the most life-threatening complication of ESLD. Immediate action is needed to protect the airway, maintain adequate perfusion and replacing both blood and clotting factors. Patients with bleeding varices require intubation and mechanical ventilation. Volume replacement can include crystalloid solutions as well as blood transfusions to attain a hemoglobin of 8 g/dL. Fresh frozen plasma (FFP) is needed to replace clotting factors that are not adequately produced in the diseased liver with the goal of an international normalized ratio (INR) of < 1.5. Platelet transfusion may be necessary to increase the platelet count to > 50 x 10⁹/L (Garcia-Tsao et al., 2007).

In order to control bleeding, an EGD may be performed to identify the vessel. If bleeding can't be controlled with banding during the EGD, balloon tamponade may be necessary. Pharmacological intervention is also necessary including splanchnic vasoconstriction with medications such as somatostatin or octreotide (Garcia-Tsao et al., 2007). Vasopressin, the most potent splanchnic vasoconstrictor, may be used, however there are numerous side effects associated with its use and patients need to be closely monitored for the development of both peripheral and cardiac ischemia (Garcia-Tsao et al., 2007). Patients who develop variceal hemorrhage are also at a higher risk of developing bacterial infections and should receive a course of prophylactic antimicrobials (Garcia-Tsao et al., 2007).

COAGULOPATHY

The liver is responsible for the production of many of the clotting factors necessary for normal coagulation. In addition, Vitamin K, a fat soluble vitamin, is needed to for the production of many components of coagulation. In ESLD, the liver may not be able to synthesize adequate clotting factors which is reflected in an elevation of the prothrombin time and INR. If there is not adequate bile production, Vitamin K may not be absorbed in sufficient amounts.

When there is active bleeding, or if there is a high risk of bleeding, FFP may be transfused to decrease the INR to < 1.5. Vitamin K may be given orally or intravenously in attempts to decrease the INR, however, if the liver is unable to produce clotting factors, no change in the INR may be seen (Kelso, 2013).

HEPATOPULMONARY SYNDROME

In patients with ESLD, changes in the pulmonary vasculature may be seen which can lead to hypoxemia. Hepatopulmonary syndrome (HPS) is not clearly understood and there are currently no ideal therapies, beyond liver transplantation, that can lead to improved oxygenation (Koch, 2014). Patients who suffer from HPS may qualify for liver transplantation even if their liver disease is not severe enough to warrant transplantation.

Additional respiratory complications can occur as a result of abdominal ascites. An hepatic hydrothorax occurs when ascitic fluid passes up from the peritoneal cavity and through the diaphragm (Baikati et al., 2014). This

is typically seen in the right pleural space and, depending upon the volume, can compromise ventilation.

HEPATORENAL SYNDROME

Hepatorenal syndrome may be seen in patients with ESLD. Decreased renal function can occur from actual damage to nephrons, can be a consequence of liver failure or can be a combination of the two. It can be difficult to differentiate between HPS and other causes of renal failure in patients with ESLD and the diagnosis of HRS is typically made when other causes of acute renal failure have been excluded (Wadei et al., 2006). Patients who have HRS typically suffer from oliguria that is not responsive to either volume or diuretics (Kelso, 2008). These patients frequently are hyponatremic and have a low urine sodium level, similar to that seen with pre-renal renal failure.

Although there are temporizing therapies that can be started, including intermittent hemodialysis or continuous renal replacement therapy, liver transplantation is the best option for patients who are eligible to be transplanted (Wadei et al., 2006). Renal function may not immediately return to normal and patients and families have to be made aware that renal replacement therapy may be needed for a period of time after receiving a liver transplant.

SUPPORT

Without a liver transplant, patients with ESLD will not survive and this can be devastating to both the patient and the family. There is a tremendous amount of psychosocial support that is needed. Not all patients are good candidates, or are eligible to receive a transplant. Any complication can be the one that leads to their death. Even those who are on the waiting list for a new liver may develop complications that will remove them from the waiting list. This then requires patient and family discussions related to the level of aggressive, life sustaining therapies and end-of-life care.

It is a difficult balancing act to maintain normal functioning for as long as possible. One small change, such as an infection, can lead a patient to lose their balance on that tightrope and end up critically ill in your ICU.

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