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Type 1 Hepatorenal Syndrome

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Introduction

Type 1 hepatorenal syndrome is kidney failure that occurs in advanced cirrhosis (Fagundes & Ginès, 2012). It is characterized by a, "significant reduction in creatinine clearance within 1-2 weeks of presentation" (Bacon, 2015). Patients that develop this condition have a median survival rate of one month (Dameron, 2011). Vasodilation leads to decreased perfusion to the kidneys and thus kidney injury (Boyer et al., 2011). Liver transplantation is the best treatment for hepatorenal syndrome (Fagundes & Ginès, 2012). Common causes of cirrhosis are hepatitis C infection, alcohol consumption, and fatty liver disease (Warner, Cuthbert, Bhore, & Rockey, 2011). Regardless of the cause of cirrhosis, the progression of hepatorenal syndrome once identified is extremely quick and deadly.

According to the National Institute of Diabetes and Digestive and Kidney Diseases (2016), cirrhosis is the twelfth leading cause of death in the United States and accounted for 36,427 deaths in 2013. In 2004, the direct costs associated with cirrhosis in the United States were $2.5 billion and the indirect costs were estimated to be $10.6 billion (Neff, Duncan, & Schiff, 2011). One estimate places the prevalence of cirrhosis in the United States at 0.27% of the population or 644,323 people (Scaglione et al., 2015). Type 2 hepatorenal syndrome differs from type 1 in that is has a slower onset and is less severe as well as less deadly. Type 1 hepatorenal syndrome is an acute condition that develops in a short period of time (within two weeks). It is important that diagnosis is timely because this condition signals likely fatality in about 30 days (Dameron, 2011). Scaglione et al. (2015) concludes that many cases of cirrhosis may be undiagnosed and, "more than half are potentially preventable by controlling diabetes, alcohol abuse, and viral hepatitis" (p. 690). Perhaps, the dissemination of information to the general public about the safety concerns related to obesity, hepatitis transmission, and alcohol use can increase self-preventative interventions and decrease deaths related to liver disease.

Cirrhosis

Though type 1 hepatorenal syndrome is an acute process, it is preceded by the chronic development of liver cirrhosis. Widespread fibrosis of the liver and the development of abnormal liver nodules is typically caused by one of three mechanisms:

- Alcoholic liver disease
- Hepatitis
- Nonalcoholic fatty liver disease

Any of these conditions can lead to cirrhosis (Perri, 2013).

The effects of advanced liver disease are many, often comorbid, and interrelated. They include:

- Portal hypertension
- Esophageal varices
- Hypersplenism
- Spontaneous bacterial peritonitis
- Hepatorenal syndrome
- Hepatic encephalopathy
- Coagulopathy
- Ascites

(Bacon, 2015)

According to Perri (2013), ascites is the main complication of cirrhosis and the mean time period to its development in liver disease is ten years. Mortality after diagnosis is estimated at fifty percent in two years. Ascites is almost always present in those that develop hepatorenal syndrome (Bacon, 2015).

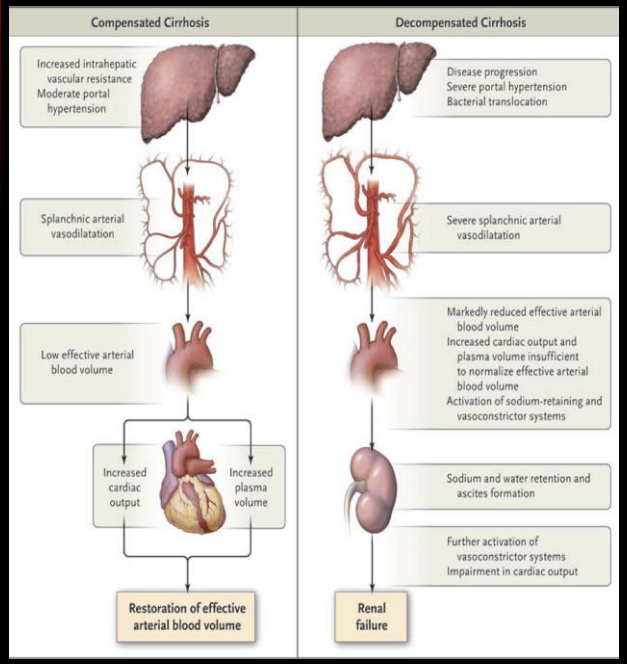

Figure 1: Compensated versus decompensated cirrhosis leading to renal failure (Fagundes & Ginès, 2012)

Pathophysiology

The best evidence suggests that the kidney dysfunction associated with type 1 hepatorenal syndrome is secondary to reduced circulatory function (Fagundes & Ginès, 2012). Cirrhosis is fibrotic scarring of the liver cells usually caused by chronic exposure to harmful agents. This sustained scarring causes reduction in vascular structure leading to a condition called portal hypertension (Møller & Bendtsen, 2015). The two factors that lead to the development of portal hypertension are increased resistance to blood flow through the liver and splanchnic vasodilation. One of the most visibly obvious symptoms associated with portal hypertension is ascites, accumulation of fluid within the peritoneal cavity. Ascites is often present in those that develop hepatorenal syndrome (Bacon, 2015). Often, the acute onset of type 1 hepatorenal syndrome is preceded by a severe infection known as spontaneous bacterial peritonitis. One third of those affected by spontaneous bacterial peritonitis will develop hepatorenal syndrome. Severe liver disease is the underlying chronic condition that leaves those affected with reduced immunity and increased susceptibility. With the presence of ascites, the peritoneal cavity proves to be a wonderful environment for bacterial proliferation (Møller & Bendtsen, 2015). Hepatorenal syndrome is characterized by increased vascular resistance in the renal circulation and reduced vascular resistance in the peripheral circulation (Bacon, 2015). This activates the renin-angiotensin-aldosterone system with even further enhancement of renal vasoconstriction and a severe imbalance of systemic and renal vasodilation (Dameron, 2011). There is no direct problem with the kidneys but rather they are being deprived of the necessary perfusion to maintain adequate function.

Process

- Exposure to agents such as hepatitis viruses and alcohol
- Liver scarring, loss of function, and loss of normal vascularity
- Increased production of nitric oxide and other vasodilating substances
- Central hypovolemia, caused by "third-spacing"
- Widespread splanchnic vasodilation and portal hypertension leading to ascites
- Spontaneous bacterial peritonitis
- Renal hypertension, peripheral hypotension, and reduced kidney perfusion
- Type 1 hepatorenal syndrome

Management and Implications for Nursing Care

Treatment of type 1 hepatorenal syndrome can include:

- Inpatient critical care setting management
- The administration of vasoconstrictors
- The administration of albumin
- Renal replacement therapy
- Transjugular intrahepatic portosystemic shunt placement
- Liver transplant

The best option for patients with type 1 hepatorenal syndrome is a liver transplant. If performed early enough, kidney function will likely be preserved (Fagundes & Ginès, 2012).

The significance of type 1 hepatorenal syndrome should not be underestimated:

- Cirrhosis is the twelfth leading cause of death in the United States (Guirguis et al., 2015)
- The costs associated with cirrhosis are in the billions annually (Neff, Duncan, & Schiff, 2011)
- Those who do not receive advanced treatment will likely die in about thirty days (Dameron, 2011)
- Most risk factors for developing this condition are preventable (Scaglione et al., 2015)

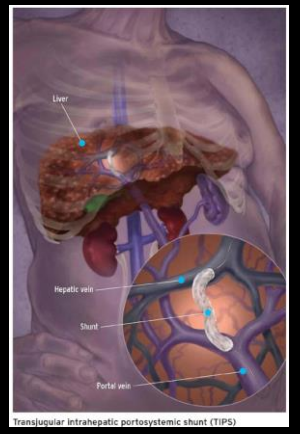

Figure 2: Transjugular intrahepatic portosystemic shunt (Dameron, 2011)

Primary prevention of type 1 hepatorenal syndrome is directly related to the prevention of liver disease. This includes vaccination for hepatitis B, safe sex practices, moderation or abstinence of alcohol intake, and diet and exercise to control weight. Secondary prevention would include reduction of alcohol intake and weight loss. Some tertiary preventative options include the use of albumin administration for those with spontaneous bacterial peritonitis and the prophylactic administration of antibiotics in patients at high risk (Fagundes & Ginès, 2012).

Conclusion

Hepatorenal syndrome type 1 is a severe and deadly manifestation of end stage liver disease. It is a direct consequence of the vasoactive effects of advanced cirrhosis such as portal hypertension, splanchnic vasodilation, and the imbalance of peripheral and renal bloodflow. Additionally, ascites with spontaneous bacterial peritonitis appears to be closely linked with the development of hepatorenal syndrome (Møller & Bendtsen, 2015). Though it is in itself an acute syndrome, the factors that precipitate the development of the condition are mostly preventable. Through educating patients about the importance of moderation in alcohol intake, receiving vaccinations, maintaining a healthy weight, among other self care practices, a reduction in preventable mortality can be achieved. There are also treatments to slow the progression and relieve the symptoms of those affected by cirrhosis.

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