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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, 2015). It is characterized by a "significant reduction in renal plasma flow" within 2 weeks of presentation (Bacon, 2015). Patients who develop this condition have a 100% fatality rate at one month (Dameron, 2013). Vasodilation leads to the development of liver disease and thus kidney injury (Bacon, et al., 2015). The best evidence suggests that the kidney dysfunction associated with type 1 hepatorenal syndrome is secondary to reduced circulating function (Fagundes & Ginès, 2012). Cirrhosis is fibrotic scarring of the liver cells usually caused by chronic exposure to harmful agents. This scarring causes the liver to "shrink", which decreases its ability to filter waste products from the blood. The presence of ascites, the periterviculumary water in the peritoneal cavity (Bacon, 2015). Severe liver disease is the "underlying chronic condition" that leaves those affected with reduced immunity due to "infiltrating leukocytes" with reduced liver function (Møller, 2015). The treatment of type 1 hepatorenal syndrome is characterized by increased renal vascular resistance in the renal cortex (Bacon, 2015). This increases the extrarenal-angiotensin aldosterone system with near enough renal vasoconstriction and a severe imbalance of systemic and renal vasodilation (Møller, 2015). There is no direct problem with the kidneys but rather they are being deprived of the necessary perfusion to maintain adequate function. Cirrhosis is the twelfth leading cause of death in the United States and accounted for 36,672 deaths in 2017. In 2019, the direct costs associated with cirrhosis in the United States were $2.5 billion and the indirect costs amounted to $10.6 billion (Neff, Duncan, & Schiff, 2011). One of the most prevalent causes of death in the United States is due to cirrhosis (Bacon, et al., 2015). The current economic burden of cirrhosis is ten years. Mortality after diagnosis is estimated at fifty percent in two years. Activity is almost always present in those with type 1 hepatorenal syndrome (Bacon, 2015).

Pathophysiology

The best evidence suggests that the kidney dysfunction associated with type 1 hepatorenal syndrome is secondary to reduced circulating function (Fagundes & Ginès, 2012). Severe liver disease is the "underlying chronic condition" that leaves those affected with reduced immunity due to "infiltrating leukocytes" with reduced liver function (Møller, 2015). The treatment of type 1 hepatorenal syndrome is characterized by increased renal vascular resistance in the renal cortex (Bacon, 2015). This increases the extrarenal-angiotensin aldosterone system with near enough renal vasoconstriction and a severe imbalance of systemic and renal vasodilation (Møller, 2015). There is no direct problem with the kidneys but rather they are being deprived of the necessary perfusion to maintain adequate function.

Cirrhosis

The effects of advanced liver disease are many, direct, controversial, and interrelated. They include:  
1. Portal hypertension  
2. Esophageal varices  
3. Hypersplenism  
4. Spontaneous bacterial peritonitis  
5. Hepatorenal syndrome  
6. Hepatomegaly  
7. Esophagus varix  
8. Features  

The best evidence suggests that the kidney dysfunction associated with type 1 hepatorenal syndrome is secondary to reduced circulating function (Fagundes & Ginès, 2012). Severe liver disease is the "underlying chronic condition" that leaves those affected with reduced immunity due to "infiltrating leukocytes" with reduced liver function (Møller, 2015). The treatment of type 1 hepatorenal syndrome is characterized by increased renal vascular resistance in the renal cortex (Bacon, 2015). This increases the extrarenal-angiotensin aldosterone system with near enough renal vasoconstriction and a severe imbalance of systemic and renal vasodilation (Møller, 2015). There is no direct problem with the kidneys but rather they are being deprived of the necessary perfusion to maintain adequate function. Cirrhosis is the twelfth leading cause of death in the United States (Ksanfomali et al., 2013). The costs associated with cirrhosis are in the billions annually (Neff, Duncan, & Schiff, 2011).

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-space”  
- Widespread splanchnic vasodilatation and portal hypertension leading to ascites  
- Spontaneous bacterial peritonitis  
- Renal hypertension, peripheral hypotension, and reduced kidney perfusion  
- Type 1 hepatorenal syndrome

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

Figure 2: Transjugular intrahepatic portosystemic shunt (Dameron, 2013)

References

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