Portal Hypertension

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Portal Hypertension
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Introduction
Chronic liver disease places the body with many significant complications. One of the more commonly identified complications is portal hypertension. In order to maintain the pressures in the portal system, increased pressures in PHT will lead to extensive complications, and eventually death. I chose to research this particular process due to the frequent presentation of it in my line of healthcare.

Pathophysiological Processes
Portal hypertension can be simply defined as increased hepatic venous pressure. Bloom, Kemp, and Luel (2015) describe PHT as “a hepatic venous pressure gradient (HVPG) greater than 5 mmHg, with complications arising to this exceeding 10 mmHg” (p. 10). The increased hepatic venous pressure is generally caused by portal blood flow resistance, which leads to various resistance constricting portal flow, which then raises higher portal pressures. This was also noted by Iwakiri, Carale, and Azer (2015). Many different liver disorders contribute to the development of portal hypertension.

Pre-hepatic Etiology
• Thrombosis
• Narrowing of the hepatic portal vein

Intra-hepatic Etiology
• Vascular remodeling
• Intrapathic shunts
• Thrombosis
• Inflammation
• Portal hypertension
• Viral hepatitis
• Schistosomiasis

Post-hepatic Etiology
• Hepatic vein thrombosis
• Right sided cardiac disorders

Underlying Pathophysiology
The underlying pathophysiology of the increased hepatic resistance is caused by “the distortion of liver architecture associated with fibrogenesis and angiogenesis, and by increased vascular hepatic tone due to intravascular coagulation secondary to an imbalance between endothelial and inflammatory mediators” (Fernandez, 2015, p.1407). The increased splanchnic flow contributes to formation of ascites, bacterial peritonitis, and portal hypertensive systemic collaterals (Fernandez, 2015). Molecular components such as decreased nitric oxide (NO), increased endothelin, monocrotaline, increased NO, and decreased microRNA, also led to increased intrarehepatic vascular resistance (IFH), thus potentiating PHT.

Breakdown of PHT:
• Increased hydrostatic pressure in portal vein causes backpressure and splanchic vasodilation
• Blood retained in dilated vessels
• Retained blood decreases renal flow and excite Renin-Angiotensin-Albumin (RAA) cascade
• Elevated Aldosterone levels try to compensate for decreased blood volume increase total body water and sodium levels
• Decreased serum albumin (diluted body water & decreased production from liver
• Decreased intravascular oncotic pressure d/d decreased serum albumin
• Fibrosis as ISF d/d increased intravascular oncotic pressure

Hepatorenal PHT Symptoms:
Portal hypertension is generally asymptomatic, but there are symptoms related to the complicated side effects of having elevated hepatic pressures:
• Varied hemorrage causing hematemesis or black tarry stools
• Mental status changes
• Abdominal distension
• Ascites
• Tenderness
• Edema
• Decreased urine output

Significance of Pathophysiology
Long term portal hypertension can be difficult to treat and lead to severe complications with life threats of severe consequences. Formation of portal hypertensive systemic community due to PHT has been identified as the major causative factor for gastrointestinal varical hemorrhage, portosystemic encephalopathy, and ascites (Fernandez, 2015). Amongst at risk for PHT are:
• Alcohol, & Mekaroonkamol (2015), “the estimated mortality for the first episode of varical hemorrhage is 30%-50%” (p. 9). The high incidence of mortality from varical hemorrhage exists as to why portal hypertension can be a significant factor for those with liver disease.

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Portal Hypertension
Portal hypertension is a complex, multifactorial complication of liver disease. Collaboration care is necessary to successfully manage and delay progression of the disease process. Quick identification of clinical signs of portal hypertension is essential to reducing mortality rates and improving quality of life for affected patients.

Implications for Nursing Care
Implications for nursing care are primarily related to assessing, monitoring, preventing, and treating complications related to PHT. The advanced nurse practitioner will monitor labs, prescribe prophylactic medications such as antisecretory agents to control blood pressures and decrease the risk of esophageal varices, as well as diuretic therapy to reduce fluid volume excess if indicated. Patient education on signs and symptoms of complications is a very important nursing implication.

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References


Additional Sources


