Portal Hypertension

Kristi Loomis
Otterbein University, kristi.loomis@otterbein.edu

Follow this and additional works at: http://digitalcommons.otterbein.edu/stu_msn

Part of the Nursing Commons

Recommended Citation
Portal Hypertension
Kristi Loomis RN, BSN
Otterbein University, Westerville, Ohio

Introduction
Chronic liver disease plagues the body with many significant complications. One of the more commonly identified complications is portal hypertension (PHT). Increased hydrostatic pressures in PHT will lead to extensive complications, and eventually death. I chose to research this pathological process due to the frequent presentation of these complications 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 hepatic venous pressure gradient (HVPG) greater than 5 mmHg, with complications arising when this exceeds 10 mmHg.” (p. 18). The increased hepatic venous pressure is generally caused by portal blood flow resistance, which leads to vasculature constriction portal flow, which in turn creates higher portal pressures. (Bloom, Kemp, & Luel, 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
• Intrap-thelial shunts
• Thrombosis
• Infarction
• Virus hepatitis
• Sclerosening

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 fibroplasia, and by increased vascular hepatic tone due to intravascular coagulation secondary to an imbalance between endogenous dilators and increased vasoconstrictor stimuli” (Fernandez, 2015, p. 1407). The increased splanchic flow contributes to formation of ascites, bacterial peritonitis, and development of portosystemic collateral veins (Fernandez, 2015). Molecular components such as decreased nitric oxide (NO), increased endothelins, microsomes, increased NOx, and decrease in microRNA also lead to increased intrathoracic vascular resistance (IVR), 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 excites Renin-Angiotensin-Aldosterone (RAA) cascade
• Elevated Aldosterone levels try to compensate for decreased blood volume increase total body water and sodium levels
• Decreased serum albumin due to dilated body water & decreased production from liver
• Decreased intravascular oncotic pressure d/t decreased albumin production
• Filtration into IFV d/t decreased intravascular oncotic pressure

Significance of Pathophysiology
Long term portal hypertension can be difficult to treat and leads to severe complications with life threats of liver and other system consequences. Formation of portosystemic collaterals circumvents the flow due to PHT has been identified as the main causative factor for gastrointestinal varical hemorrhage, portosystemic encephalopathy, and sepsis (Fernandez, 2015). Arising out of PHT, Auer, & Mekarskamro (2015), “the estimated mortality rate for the first episode of varical hemorrhage is 30-50%”. (p. 9). The high incidence of mortality from varical hemorrhage tends evidence as to why portal hypertension is a significant factor for those with liver disease. Early intervention and disease surveillance is of primary concern when dealing with PHT.

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 vasoactive agents to control blood pressures and decrease the risk of esophageal varices, as well as dinitic therapy to reduce fluid volume excess if induced. Patient education on signs and symptoms of complications is a very important nursing implication. Nurse practitioners will order studies such as liver and abdominal ultrasound, and perform or refer testing to assess the identification of portal hypertension. The nurse practitioner will also collaborate with other specialists to manage the disease process. Gastroenterology and radiology specialists perform lifesaving procedures such as endoscopic sclerotherapy to stop bleeding varices, or transjugular intrahepatic portosystemic shunt (TIPS) procedures which help to reduce portal pressure.

<table>
<thead>
<tr>
<th>Patient Education Tips:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monitor for Sx of bleeding</td>
</tr>
<tr>
<td>Monitor platelet counts &amp; INR</td>
</tr>
<tr>
<td>Monitor for Sx of ascites (i.e. Increased abdominal girth, bulging flanks, abdominal fluid wave)</td>
</tr>
</tbody>
</table>

References

Additional Sources