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Portal Hypertension

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Introduction

Chronic liver disease plagues the body with many significant complications. One of the more commonly identified complications is portal hypertension (PHT). Without intervention the elevated pressures in PHT will lead to extensive complications, and eventually death. I chose to research this pathological process due to the frequent presentation of PHT related complications in my line of healthcare.

Pathophysiological Processes

Portal hypertension can be simply defined as increased hepatic venous pressure. Bloom, Kemp, and Lubel (2015) describe PHT "as hepatic venous pressure gradient (HVPG) greater than 5 mmHg, with complications arising once this exceeds 10 mmHg" (p. 16). The increased hepatic venous pressure is generally caused by portal blood flow resistance, which leads to vascular resistance constraining portal flow, which then creates higher portal pressures, also known as PHT (Iwakiri, 2014). 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
- Intrahepatic shunts
- Thrombosis
- Inflammation
- Fibrosis/cirrhosis
- Viral hepatitis
- Schistosomiasis

Post-hepatic Etiology:

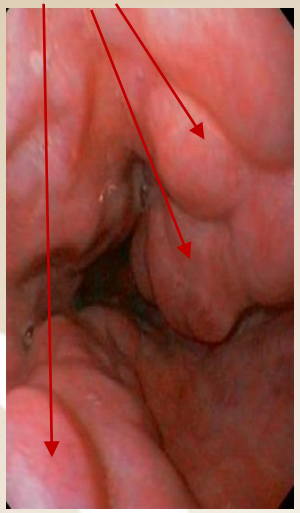
- Hepatic vein thrombosis
- Right sided cardiac disorders

Portal hypertension causes many clinically identifiable complications:

Long-term PHT Signs

- Esophageal & Gastric Varices (See image)
- Splenomegaly
- Ascites
- Jaundice
- Sepsis syndrome
- Caput Medusae
- Portosystemic encephalopathy

Esophageal Varices



Esophageal Varices [Online image]. Retrieved June 28, 2016 from http://www.endoatlas.org/assets/media/img/xl/weo_eso_varicesc_costam.jpg

Hepatorenal PHT Symptoms:

Portal hypertension is generally asymptomatic, but there are symptoms related to the complicated side effects of having elevated hepatic pressures:

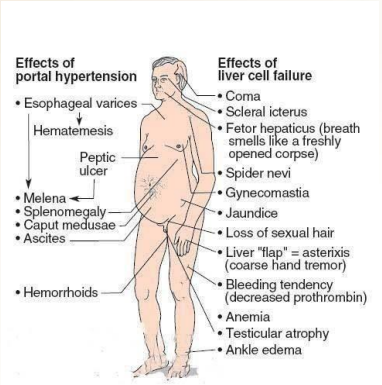
- Variceal hemorrhage causing hematemesis or black tarry stools
- Mental status changes
- Abdominal distention
- Asterixis
- Tiredness
- Edema
- Decreased Urine Output

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 intrahepatic vasoconstriction secondary to an imbalance between decreased endogenous dilators and increased vasoconstrictor stimuli" (Fernandez, 2015, p.1407). "Portal Hypertension is aggravated by an augmented blood flow in the splanchnic organs draining into the portal vein due to enhanced vasodilation and angiogenesis, and subsequent elevation in portal venous inflow which significantly affects the progression of PHT" (Fernandez, 2015, p.1407). The increased splanchnic flow contributes to formation of ascites, bacterial peritonitis, and portosystemic collateral vessels (Fernandez, 2015). Molecular components such as decreased nitric oxide (NO), increased endothelin, eicosanoids, increased nogo-B, and decrease in microRNAs, also lead to increased intrahepatic vascular resistance (IHVR), thus potentiating PHT.

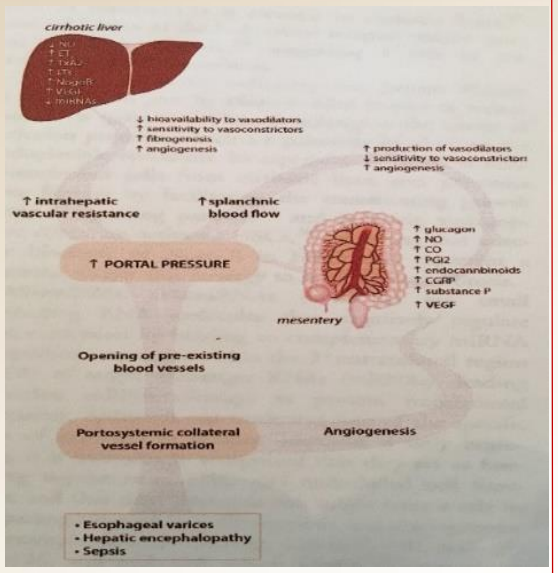
Breakdown of PHT:

- Increased hydrostatic pressure in portal vein causes backpressure and splanchnic 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 d/t diluted body water & decreased production from liver
- Decreased intravascular oncotic pressure d/t decreased serum albumin
- Filtration into ISF d/t decreased intravascular oncotic pressure

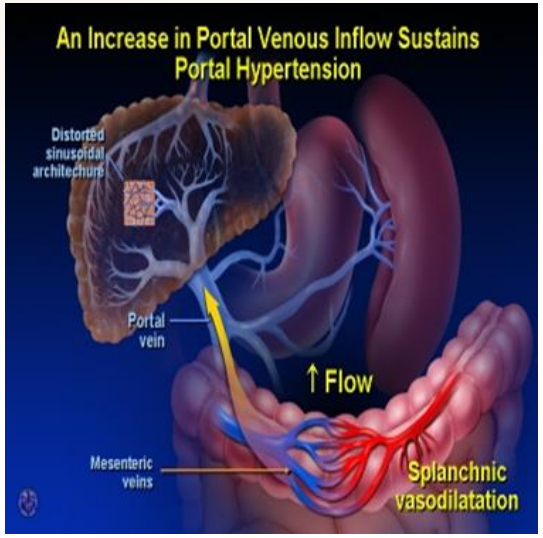


Presentation of cirrhosis/portal hypertension.

Portal Hypertension [Online image]. Retrieved June 28, 2016 from http://media-cache-ec0.pinimg.com/736x/f4/ce/98/f4ce98e442d41d96f5c1e64e33091b41.jpg



Fernandez, M. (2015). Molecular pathophysiology of portal hypertension [Untitled illustration]. *Hepatology*, *61*(4), 1406-1415. doi:10.1002/hep.27343



Portal Blood Flow [Online image]. Retrieved June 28, 2016 from http://www.gastroslides.org/media/10125.jpg

Significance of Pathophysiology

Long term portal hypertension can be difficult to treat and leads to severe complications with life threatening consequences. Formation of portosystemic collateral vessels due to PHT has been identified as the main causative factor for gastroesophageal variceal hemorrhage, portosystemic encephalopathy, and sepsis (Fernandez, 2015). According to Carale, Azer, & Mekaroonkamol (2015), "the estimated mortality for the first episode of variceal hemorrhage is 30-50%" (p. 9). The high incidence of mortality from variceal hemorrhage lends 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 diuretic therapy to reduce fluid volume excess if indicated. Patient education on signs and symptoms of complications is a very important nursing implication.
Nurse practitioners will order studies such as liver and abdominal ultrasounds, and barium swallow testing to assist in the identification of portal hypertension. The nurse practitioner will also as collaborate with other specialists to manage the disease process. Gastrointestinal and radiology specialist perform lifesaving procedures such as endoscopic variceal band ligation (EVBL) to stop bleeding varices, or transjugular intrahepatic portosystemic shunt (TIPS) procedure which helps to reduce portal pressure.

Patient Education Tips:

- Monitor for S/S of bleeding
- Monitor platelet counts & INR
- Monitor for S/S of ascites (i.e. Increased abdominal girth, bulging flanks, abdominal fluid wave)

Conclusion

Portal hypertension is a complex, multifactorial complication of liver disease. Collaborative 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 living for affected patients. Addressing and treating the underlying cause of PHT is paramount for long-term survival. Failure to effectively treat PHT will have disastrous consequences, ultimately leading to death.

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Additional Sources

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