



## Portal Hypertension in Adults- A Comprehensive Review

Authors

**Sweta.V.R, Dr Naufal Rizwan**

Department of General Medicine, Saveetha Medical College, Chennai

### ABSTRACT

**AIM:** This article aims at providing an overview regarding Portal hypertension in adults.

**OBJECTIVE:** To provide a comprehensive review on portal hypertension in adults.

**BACKGROUND:** Portal hypertension is a clinical condition wherein the pressure inside the portal venous system is increased. Portal venous pressure is usually calculated by measuring the hepatic venous pressure gradient (HVPG), and portal hypertension is said to be present when HVPG is greater than 5mm Hg. The major causes of portal hypertension can be classified as pre-hepatic, hepatic and post hepatic causes. The presence of portal hypertension in an adult can lead to various complications such as ascites, hepato-renal syndrome, hepatic encephalopathy, development of oesophago-gastric varices etc. this article aims at providing an overview about the various causes, symptoms and treatment of portal hypertension.

**REASON:** To provide an updated comprehensive review on portal hypertension in adults.

**KEY WORDS:** hepatic venous pressure gradient, ascites, hepato-renal syndrome, hepatic encephalopathy, oesophago-gastric varices.

### INTRODUCTION

Portal hypertension is a clinical syndrome characterized by a hepatic venous pressure gradient (HVPG) exceeding 5 mmHg. Hepatic venous pressure gradient is the difference between portal pressure and inferior vena cava pressure. Portal hypertension can present as oesophago-variceal bleeding, ascites or hypersplenism.<sup>1</sup> Portal hypertension is initiated by increased outflow resistance; this can occur at a pre-sinusoidal (intra- or extra hepatic), sinusoidal, or post-sinusoidal level. As the condition progresses, there is a rise in portal blood flow, a combination that maintains and worsens the portal hypertension<sup>2</sup>. Portal hypertension results in collateral circulation between the systemic circulation and portal circulation. The commonest

sites of collateral circulation are distal oesophagus, stomach, rectum, umbilicus and retroperitoneum<sup>3</sup>.

### CLASSIFICATION

Classification of portal hypertension<sup>3</sup>:

#### -Prehepatic:

Portal vein thrombosis

Splenic vein thrombosis

#### -Hepatic:

\*Pre-sinusoidal:

Schistosomiasis

Primary biliary cirrhosis

Idiopathic portal hypertension

Non-cirrhotic portal fibrosis

\*Sinusoidal:

Alcoholic cirrhosis

Cryptogenic cirrhosis  
 Post necrotic cirrhosis  
 Alcoholic hepatitis  
 \*Post-sinusoidal  
 Veno-occlusive disease  
**-Posthepatic:**  
 Budd chiari syndrome  
 IVC obstruction  
 Constrictive pericarditis.

## ETIOLOGY

In a survey done to study the etiology of portal hypertension in adults in South India<sup>1</sup>, it was observed that after the initial work-up (prior to liver biopsy) the commonest etiology for portal hypertension was cryptogenic chronic liver disease, followed by alcohol, hepatitis B and HCV related chronic liver disease. Other etiologies were portal vein thrombosis, with or without associated cryptogenic chronic liver disease, Budd Chiari syndrome, Autoimmune liver disease, biliary etiology, Wilson's disease, cardiac cirrhosis. Twenty four patients had >1 etiology for chronic liver disease and portal hypertension: hepatitis B and alcohol; hepatitis C and alcohol; 25 patients had hepato-cellular carcinoma. Alcohol intake in India is steadily increasing, with decrease in the initiation age. This alarming trend is noticed in many areas of the country<sup>4,5</sup>. Alcohol is a significant contributor to the increase in occurrence of portal hypertension amongst the Indian population.

## DIAGNOSIS

Presence of varices, variceal hemorrhage or ascites indicates presence of portal hypertension. The assessment of HVPG (hepatic venous pressure gradient) and endoscopy for the assessment of oesophageal varices is sufficient for the diagnosis of clinically significant portal hypertension. Variceal pressure measurement and Doppler-ultrasound seem promising but, due to inter equipment and inter-observer variability, their use in clinical practice cannot be recommended<sup>6</sup>. For the presence of gastric varices the classification of Sarin et al should be used<sup>7</sup>.

For the detection of fundal varices, presence of red signs, large size and Child class B or C should be considered as risk factors for bleeding<sup>6</sup>.

## SYMPTOMS

Portal hypertension is detected by the presence of one or more of the three main symptoms- variceal hemorrhage, ascites and hepatic encephalopathy.

### Formation of varices:

When the portal pressure gradient increases, it is decompressed by diverting 90% of its flow back to the heart resulting in remodeling and enlargement of the portal vessels. VEGF,NO-driven VEGF type II receptor expression and PDGF drive this process<sup>8,9</sup>. A common location for such vessels is at the gastroesophageal junction at which they lie immediately subjacent to the mucosa and present as gastric and esophageal varices. Varices do not form until the HVPG exceeds 10 mm Hg and usually do not bleed unless the HVPG exceeds 12mm Hg<sup>10,11</sup>. Variceal hemorrhage is determined by high portal pressure and variceal diameter. Varices are most superficial in the gastro-esophageal junction and hence has the thinnest wall in that region. Therefore oesophageal variceal hemorrhages occurs mostly in this region. HPVG>20mm Hg has been associated with continued bleeding and failure of medical therapy<sup>12</sup>.

### Ascites

Ascites is a common complication of cirrhosis<sup>13</sup>. The pathophysiological processes that result in ascites are- systemic arteriolar vasodilatation, activation of Na and H<sub>2</sub>O retention, and sinusoidal portal hypertension.

### Hepatic Encephalopathy

Hepatic encephalopathy (HE) is that which encompasses mental status changes in subjects with acute and chronic liver failure. Variable degrees of hepatocellular failure and portal-systemic shunting can produce HE. Ammonia is a key factor in the pathogenesis of HE<sup>14</sup>. Infection, which promotes inflammation, can precipitate HE.<sup>15,16</sup>

## MANAGEMENT OF PORTAL HYPERTENSION

Management of portal hypertension is done by managing its symptoms- variceal hemorrhage, ascites and hepatic encephalopathy.

### Management of Variceal Hemorrhage

Variceal bleeding occurs when the HVPG is greater than 12mm Hg. When the patient has medium to large varices along with Child-Pugh class B or C cirrhosis and varices of any size are considered to be at high risk<sup>17</sup>. Non selective  $\beta$ -blockers produce vasoconstriction and thereby decrease the portal pressure. Endoscopic variceal ligation (EVL) reduces the risk of bleeding and is used for patients who have an intolerance for  $\beta$  blockers. For those patients who have rebleeding, packed red cells are transfused to maintain the blood hemoglobin level at 9gm/dl. Balloon tamponade can effectively produce temporary hemostasis in 80%–90% of cases.<sup>18,19</sup> Transjugular intrahepatic portosystemic shunts (TIPS), a radiologic procedure by which a tract is created between the hepatic and portal vein and kept open by deployment of a coated stent, is the salvage procedure of choice in most subjects.<sup>20-22</sup> TIPS produces hemostasis in over 90% of cases and is effective both for gastric and esophageal variceal bleeding.<sup>22</sup>

### Management of Ascites

Spironolactone inhibits distal tubular Na reabsorption by antagonizing aldosterone. The biologic effect half-life of spironolactone extends over days. It can therefore be dosed once a day, and dose changes should not be performed at less than 7-day intervals<sup>23</sup>. Large volume paracentesis (5 L removed at a single sitting) (LVP) is used mainly for symptom relief and rapid mobilization of tense ascites.<sup>24</sup> Sodium reduction is a mandatory step towards management of ascites

### Management of Hepatic Encephalopathy

Neomycin, metronidazole, and rifaximin, which have widely different antimicrobial spectra, have been used to treat HE. Flumazenil, a benzodiazepine receptor antagonist, indicated a

beneficial effect on short-term awakening from deeper stages of encephalopathy<sup>25</sup>.

## CONCLUSION

Thus in conclusion, Portal hypertension is a clinical syndrome wherein the hepatic venous pressure gradient (HVPG) is more than 5 mmHg. The commonest causes of portal hypertension in India are cryptogenic chronic liver disease, followed by alcohol, hepatitis B and HCV related chronic liver disease. The main complications caused by portal hypertension are variceal bleeding, ascites and hepatic encephalopathy. Patients with severe persistent gastro-intestinal hemorrhage have higher morbidity and mortality rate. The risk of death is maximal during the first few days after the variceal bleeding episode and decreases slowly over the first 6 weeks. However, despite improvements in therapy, the mortality rate at 6 weeks remains greater than 20%; this rate is higher when surgical intervention is needed.<sup>26</sup>. Patients with a hepatic venous pressure gradient (HVPG) of 20 mm Hg measured 24 hours after the onset of bleeding esophageal varices have a higher 1-year mortality rate<sup>27</sup>.

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