

Non-cirrhotic Portal Hypertension: Not Every Portal Hypertension Indicates Cirrhosis

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Description

Portal hypertension is a medical condition characterized by increased pressure within the portal venous system, a network of veins that carries blood from the digestive organs to the liver. Its most commonly associated with liver cirrhosis, a condition where the liver becomes scarred and dysfunctional due to chronic injury or inflammation. However, portal hypertension can also occur in non-cirrhotic conditions. This article explores the concept of non-cirrhotic portal hypertension, its causes, clinical implications, and management. To understand non-cirrhotic portal hypertension, we must first grasp the basics of portal hypertension. The portal venous system plays a crucial role in the metabolism and filtration of blood from the digestive organs. When blood flows through the liver, it encounters resistance due to the intricate network of blood vessels within the liver tissue. This resistance is essential for the liver to perform its functions of detoxification, metabolism, and protein synthesis [1]. In cirrhosis, chronic liver injury leads to the formation of fibrous tissue, which disrupts the liver's architecture and impedes blood flow. This results in increased pressure in the portal venous system, causing a host of complications.

Cirrhotic portal hypertension is the most common cause of elevated portal pressure. It occurs due to the scarring of the liver tissue, leading to architectural changes in the liver. The scarring disrupts the normal blood flow through the liver, increasing pressure within the portal venous system. Portal hypertension causes the development of collateral blood vessels, known as varices, in the oesophagus and stomach. These varices are prone to rupture, leading to life-threatening bleeding. The increased portal pressure causes fluid to leak into the abdominal cavity, resulting in ascites, a condition characterized by abdominal swelling and discomfort. Portal hypertension can lead to the accumulation of toxic substances in the blood, causing cognitive impairment and other neurological symptoms. Enlargement of the spleen is a common finding in portal hypertension due to congestion of blood in the splenic vein [2].

While cirrhosis is the most well-known cause of portal hypertension, it's crucial to recognize that this condition can occur in the absence of cirrhosis. Non-cirrhotic portal hypertension, also known as NCPH, refers to portal hypertension that arises from conditions other than liver cirrhosis. It is essential to understand the diverse etiologies and presentations of NCPH to provide appropriate management and care. Idiopathic Portal Hypertension (IPH) is a term used when the cause of portal hypertension is unknown. It is a diagnosis of exclusion, made when other known causes have been ruled out. The exact mechanisms underlying IPH are not well understood. Some conditions can increase portal pressure by obstructing blood flow before it reaches the liver [3]. Examples include portal vein thrombosis and extra hepatic portal vein obstruction. Conditions that primarily affect the liver but do not lead to cirrhosis

can also cause portal hypertension. These include hepatic fibrosis, nodular regenerative hyperplasia, and sinusoidal obstruction syndrome. Certain conditions can obstruct blood flow after it leaves the liver, causing increased portal pressure. Budd-chiari syndrome, which involves the blockage of hepatic veins, is an example of a post hepatic cause of portal hypertension.

The clinical features of non-cirrhotic portal hypertension can vary depending on the underlying cause. However, some common manifestations include enlargement of the spleen is a hallmark of portal hypertension, irrespective of the cause. This can lead to symptoms such as left upper quadrant abdominal pain and early satiety. Patients with non-cirrhotic portal hypertension may also develop varices in the oesophagus and stomach, making them susceptible to variceal bleeding. While ascites is more commonly associated with cirrhotic portal hypertension, it can also occur in NCPH, particularly in patients with hepatic vein obstruction (Budd-Chiari syndrome). Bleeding from varices, whether due to cirrhotic or non-cirrhotic portal hypertension, is a serious complication that can result in hematemesis (vomiting of blood) and melena (black, tarry stools). This condition, characterized by confusion, altered consciousness, and neuromuscular dysfunction, can occur in both cirrhotic and non-cirrhotic portal hypertension due to the shunting of toxic substances around the liver.

The diagnosis of non-cirrhotic portal hypertension requires a comprehensive approach that involves medical history, physical examination, and various diagnostic tests. Here are some key steps in the diagnostic process: A thorough medical history is crucial to identify potential risk factors, underlying conditions, and symptoms. Physical examination may reveal signs such as splenomegaly, ascites, or signs of gastrointestinal bleeding. Blood tests can help assess liver function and identify any abnormalities, such as elevated liver enzymes or reduced platelet counts. In non-cirrhotic portal hypertension, liver function may be relatively preserved compared to cirrhotic patients. Imaging plays a significant role in diagnosing non-cirrhotic portal hypertension and identifying its underlying cause. Ultrasound, Computed Tomography (CT), and Magnetic Resonance Imaging (MRI) can provide valuable information about the liver, spleen, and blood flow within the portal venous system. Upper endoscopy is essential to assess the presence of esophageal or gastric varices, which are common in portal hypertension. In some cases, a liver biopsy may be necessary to evaluate liver tissue and confirm the absence of cirrhosis or to diagnose specific liver conditions associated with NCPH [4].

The management of non-cirrhotic portal hypertension depends on the underlying cause and the severity of the condition. Here are some general principles of management. The first step is to identify and address the specific condition causing portal hypertension. For example, anticoagulation therapy may be used to treat portal vein thrombosis, while angioplasty or stent placement may be necessary for Budd-Chiari syndrome. Medications can be used to manage symptoms and complications. Beta-blockers are often prescribed to reduce portal pressure and prevent bleeding from varices. Lactulose or other medications may be used to manage hepatic encephalopathy [5].

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Conflict of Interest

There are no conflicts of interest by author.

References

1. Schouten, Jeffrey NL, Frederik Nevens, Bettina Hansen and Wim Laleman, et al. "Idiopathic noncirrhotic portal hypertension is associated with poor survival: Results of a long-term cohort study." *Aliment Pharmacol Ther* 35 (2012): 1424–1433.
2. Siramolpiwat, Sith, Susana Seijo, Rosa Miquel and Annalisa Berzigotti, et al. "Idiopathic portal hypertension: Natural history and long-term outcome." *Hepatology* 59 (2014): 2276–2285.
3. Barge, Sandrine, Veronique Grando, Jean-Charles Nault and Chole Broudin, et al. "Prevalence and clinical significance of nodular regenerative hyperplasia in liver biopsies." *Liver Int* 36 (2016): 1059–1066.
4. Guido, Maria, Venancio AF Alves, Charles Balabaud and Prithi S. Bathal, et al. "International Liver Pathology Study Group. Histology of portal vascular changes associated with idiopathic non-cirrhotic portal hypertension: Nomenclature and definition." *Histopathology* 74 (2019): 219–226.
5. Mahamid, Jamal, Ines Miselevich, Dina Attias and Ruth Laor, et al. "Nodular regenerative hyperplasia associated with idiopathic thrombocytopenic purpura in a young girl: A case report and review of the literature." *J Pediatr Gastroenterol Nutr* 41 (2005): 251–255.

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