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Imaging diagnosis of portal hypertension☆

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Abstract Portal hypertension (PHT) is a clinical entity defined when hydrostatic pressure >5 mm Hg in the portal venous territory, being clinically significant if it reaches ≥10 mm Hg. At this threshold, complications can develop, such as the bleeding of esophageal varices, the appearance of ascites, or hepatic encephalopathy. Imaging modalities play an important role here as non-invasive methods to determining whether PHT is present or not. This article analyzes various imaging findings that can be suggestive of PHT and contributes to define its etiology, severity, and possible complications.

PALABRAS CLAVE
Hipertensión portal;
Ecografía Doppler;
Circulación colateral;
Venas coronarias

Diagnóstico por imagen de la hipertensión portal

Resumen La hipertensión portal (HTP) es una condición clínica definida por una presión hidrostática >5 mmHg en el territorio venoso portal, siendo clínicamente significativa cuando es ≥10 mmHg. A partir de este umbral pueden desarrollarse complicaciones, como sangrado de varices esofágicas, aparición de ascitis o encefalopatía hepática. Las técnicas de imagen tienen un papel importante como método no invasivo para determinar la presencia de HTP. En este artículo se analizan varios hallazgos radiológicos que pueden sugerir HTP y contribuir a definir su etiología, gravedad y posibles complicaciones.

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Introduction

Portal hypertension (PHT) is a well-defined clinical condition consisting of hydrostatic pressure in the venous portal venous territory >5 mmHg (normal pressures are between 1 and 5 mmHg). It is not considered clinically significant until hydrostatic pressure reaches ≥10 mmHg, a threshold from which associated clinical complications can occur: bleeding of esophageal varices, ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, and hepatopulmonary or hepatorenal syndrome.[1–4]

The increased pressure in the portal venous system is initially due to increased intrahepatic vascular resistance, whether prehepatic or posthepatic, followed by increased splanchnic flow. All this determines the formation or restoration of a network of venous collateral blood vessels destined to decompress the system and redirect part of the portal flow toward systemic circulation while avoiding the liver.[2,5]

This increased pressure can have different causes, being hepatic cirrhosis the most common cause in our setting.[6,7]

This paper analyses various radiological findings, specially ultrasound findings, suggestive of the presence of PHT that allow us to define its etiology, severity, and possible complications.

Diagnosis

The reference pattern to determine portal venous pressure is to measure the portal pressure gradient through the catheterization of suprahepatic veins (SHV). Its invasive nature and limited availability have led to the development of non-invasive alternatives for the diagnosis of PHT, among which we have the mention here the Doppler ultrasound, the elastography techniques, and certain analytical parameters.

Thrombocytopenia is one of the analytical parameters that keeps a narrower correlation with the portal pressure gradient. Similarly, one model combining albumin, alanine aminotransferase, and the INR (International Normalized Ratio) has also proven effective to predict portal hypertension in compensated cirrhosis.[8]

Today, elastography is a non-invasive method of great clinical utility. There are different techniques available today, such as Transient Elastography (TE) or Fibroscan, the Acoustic Radiation Force Impulse Imaging (ARFI), or the Real-time Shear Wave Elastography (SWE). These imaging modalities add structural information to the morphological properties that the ultrasound scan or the magnetic resonance imaging (MRI) provide by assessing hepatic elasticity, thus being the closest diagnostic alternative to hepatic biopsy for the diagnosis of patients with cirrhosis. Several studies have confirmed its excellent correlation with the portal pressure gradient.[9–11] All this has made elastography an essential tool to be able to make decisions on the best therapeutic approach for the patient, including screening for hepatocellular carcinoma and esophageal varices.[12,13]

The B-mode Doppler ultrasound still plays a crucial role as a non-invasive alternative, since it can show the presence of PHT, define its etiology, severity, and possible complications. Also, it is useful for the follow-up of patients who have undergone transjugular intrahepatic portosystemic shunt (TIPS) procedures.[14,15]

Table 1 Radiological findings of portal hypertension.

Portal caliber >13 mm
Splenomegaly
Doppler US:
Reduced portal velocity (<15 cm/s) or hepatofugal flow
Increased arterial RI (>0.7)
Portosystemic collaterality
Ascites

Radiological findings in the diagnosis of portal hypertension

The Doppler ultrasound is the most advanced imaging modality for the study of PHT due to its accessibility, safety and good cost-effectiveness ratio. Several ultrasound findings have been proposed as markers of PHT, both morphological and hemodynamic (Table 1 and Fig. 1). The utility of some of these findings has been put into question by different studies, mainly due to the high inter- and intraobserver variability of ultrasound measurements.

- Portal caliber

Portal calibers >13 mm suggest PHT with 100% specificity and an approximate 40% sensitivity[15–18] due to cases of PHT with portosystemic collateral shunts. The main setback of

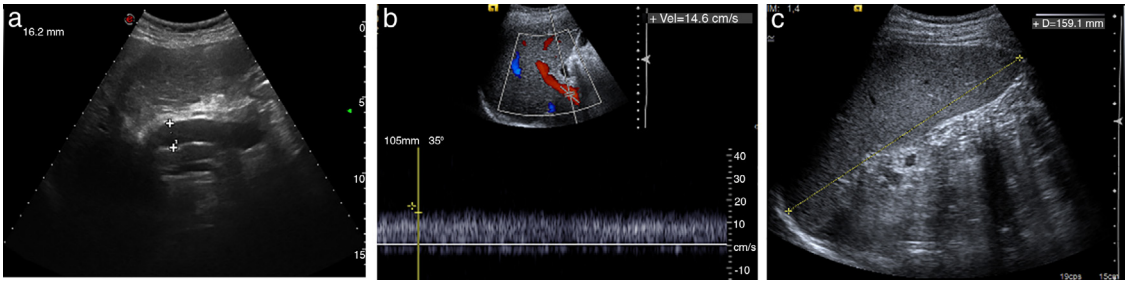

Figure 1 Seventy-three-year-old patient on ultrasound follow-up due to hepatic cirrhosis and portal hypertension. The ultrasound longitudinal plane on the portal vein (a) shows an increased portal caliber (16 mm). The Doppler spectrum conducted at hepatic hilum level on one right intercostal plane (b) shows reduced portal velocity (14.6 cm/s). Finally, one longitudinal plane on the spleen (c) shows one 16 cm-splenomegaly.