MORPHOLOGICAL HEPATIC ALTERATIONS OF CIRRHOSIS: IMAGING FINDINGS

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Resumen

La cirrosis es una causa importante de morbilidad y mortalidad en el mundo. El diagnóstico de cirrosis es tradicionalmente establecido con resultados de biopsia, pero puede ser sugerido por los cambios morfológicos visualizados en imágenes. Su reconocimiento es esencial para la caracterización de las lesiones focales hepáticas. El objetivo de este trabajo es reconocer los cambios morfológicos visualizados en imágenes en la cirrosis hepática.

La cirrosis es comúnmente causada por la hepatitis crónica de origen infeccioso o el abuso de alcohol, si bien un gran número de desórdenes que causan lesión hepática pueden llevar al desarrollo de esta entidad. Patológicamente se define por tres características principales: fibrosis, transformación nodular y distorsión de la arquitectura hepática.

Abstract

Cirrhosis is a major cause of morbidity and mortality worldwide. The diagnosis of cirrhosis is traditionally established through biopsy results, but it may be suggested by morphological changes displayed in images. Its recognition is essential for the characterization of focal liver lesions. The aim of this paper is to recognize the morphological changes observed in images of cirrhosis. Cirrhosis is commonly caused by chronic hepatitis due to an infection or alcohol abuse, although a large number of disorders that cause liver injuries may lead to the development of this condition. Pathologically it is defined by three main features: fibrosis, nodular transformation and distortion of hepatic architecture. Subtle morphological changes of the liver may be the earliest detectable signs in images, including the enlargement of the hilar periportal space, the interlobar

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Sutiles cambios morfológicos del hígado pueden ser los primeros signos detectables en imágenes, incluyendo la ampliación del espacio periportal hiliar, de la cisura mayor interlobar y el agrandamiento de la fosa vesicular. Otros signos típicos son la atrofia del segmento medial del lóbulo izquierdo y del segmento anterior del lóbulo derecho o la hipertrofia del lóbulo caudado y del segmento lateral del lóbulo izquierdo. La nodularidad del borde hepático es otra característica visualizada en la cirrosis y está relacionada con la presencia de nódulos de regeneración. También se pueden ver signos derivados de la hipertensión portal, incluyendo colaterales venosas, esplenomegalia, ascitis, entre otros.

**Palabras clave:** Cirrosis, alteraciones morfológicas.

**Key words:** Cirrhosis, morphological alterations.

**Introduction**

Hepatic cirrhosis is one of the main health problems in the world due to its high morbidity and mortality. It is a chronic and irreversible liver disease that appears in the final stages of diverse pathologies. The cellular lesion triggers an inflammation, regeneration and fibrosis cycle that leads to an alteration of the intrahepatic circulation, portal hypertension and cholestasis.

In anatomic pathology examinations, it is characterized by an extensive fibrosis and the presence of numerous regeneration nodules. Depending on the size of these nodules, cirrhosis can be classified as micronodular (smaller than 3 mm), macronodular (bigger than 3 mm) and mixed (1).

Among the most common causes of cirrhosis, there is excessive alcohol consumption and hepatitis B or C virus infection. Less frequent causes include chronic hemochromatosis, biliary obstruction and hepatic congestion, use of pharmaceuticals and toxins, and hereditary disorders, such as Wilson’s disease, alpha 1-antitrypsin deficiency and glycogenosis type IV.

In images studies, cirrhosis is characterized by alterations in the morphology, in the edges of the liver, and in the parenchyma, with regeneration nodules and fibrosis. There are also extrahepatic manifestations such as the development of portosystemic collaterals, ascites and splenomegaly (2).

The main role of the radiologist consists of evaluating the size of the liver and of its diverse segments, perform a biometric analysis of the segments I and IV in search of early signs of cirrhosis, analyze hepatic edges exhaustively, and identify the effects of portal hypertension. The presence of focal lesions in a cirrhotic liver must be interpreted as hepatocellular carcinoma in a first diagnosis but focal lesions in liver without chronic pathology determine differential diagnosis.
Analysis of the hepatic edge and parenchyma

The margins of the hepatic gland must be smooth, but cirrhosis often makes them nodular due to the existence of numerous regeneration nodules. The aspect of the hepatic edge will depend on the size of these regeneration nodules. The edge can be nodular and thin in cases of micronodular cirrhosis (Figure 1) or nodular and thick in cases of macronodular cirrhosis (Figure 2). Hepatic regeneration nodules are isodense with the glandular parenchyma in CT and isointense in T1 and T2-weighted images in MRI. Sometimes, they can be hyperattenuating in CT images without contrast and hypointense in T1 and T2 sequences of MRI due to the presence of hemosiderin (siderotic nodules).

A smaller amount of cirrhotic livers has a heterogeneous parenchyma in CT or MRI (2). The main causes of this heterogeneity are the presence of fibrosis, irregular fatty liver and iron deposits. Hepatic fibrosis is hypodense in relation to the parenchyma in CT without intravenous contrast and can show a late enhancement after the administration of the contrast agent (Figure 3). In MRI, it is often hypointense in T1-weighted sequences and hyperintense in T2-weighted sequences.

Fibrosis can adopt several morphological patterns. It can be patchy, thin, appear as thick perilobular bands and/or perivascular cuffing producing an ox eye pattern. An irregular fatty liver produces patchy areas of less density in CT and it is frequent in patients with alcoholic liver cirrhosis who are still drinking alcohol (2). When the heterogeneity of the parenchyma is caused by iron deposits, there are areas of high density in CT without contrast that become hypointense in MRI in T2-weighted sequences.

Signs of dimorphism: hepatic atrophy and hypertrophy

Approximately 25% of cirrhotic livers in the final stage have a normal size. 36% presents diffuse atrophy and the rest of the patients present a combination of segmental atrophy and hypertrophy.

An early sign of cirrhosis is the increase of hilar periportal space, which is filled with fat content due to the atrophy of segment IV. Normally, the hilar periportal space measures less than 10 mm from the anterior edge of the right portal branch to the posterior edge of the medial segment of the left lobe. A size greater that 10 mm represents a sensitivity of 93% and a specificity of 92% for the diagnosis of cirrhosis (3) (Figure 4).

Focal atrophy is more common in the right hepatic lobe and in the medial segment of the left hepatic lobe (Figure 5). A sign produced by atrophy, which is very specific of cirrhosis, is the presence of a clear groove in the right posterior surface of the liver. This groove is called posterior hepatic notch and it represents a sensitivity of 72% and a specificity of 98% for the diagnosis of cirrhosis (Figure 6).

The segments that present hypertrophy with a greater frequency are the caudate lobe and the lateral segments of the left hepatic lobe. Biometry of segment I has proved to be useful for the evaluation of the relationship between segment I and the right lobe of the liver. If this relationship is greater than 0.9, there is a sensitivity of 71.1% and a specificity of 77% for the diagnosis of cirrhosis. Biometry of segment I is performed by drawing three vertical lines: one through the internal edge of segment I, another one through the external edge of the left liver and a last imaginary line that goes through the right lateral wall of the bifurcation of the main portal vein, parallel to the middle sagittal plane of the body.

Portosystemic venous collaterals: signs of portal hypertension

In patients with cirrhosis and portal hypertension, part of the portal venous flow reverts its direction to the systemic circulation through portosystemic anastomosis. From the clinical point of view, the most important venous collaterals are esophageal and paraesophageal varices, due to the risk of digestive hemorrhage. Esophageal varices are dilated veins located in the wall of the lower part of the esophagus, while paraesophageal varices are located outside the digestive wall (Figure 7). Both receive the flow from the left gastric vein, which is divided into anterior branches to supply esophageal varices and posterior branches to supply paraesophageal varices.

Another type of portosystemic shunts is that which communicate the spleno-portal axis with the
left renal vein, through branches of the splenic vein (spleno-renal) and the coronal vein (gastro-renal). They are seen with CT as tortuous and dilated venous structures in the retroperitoneum that drain into an ectatic left renal vein. The paraumbilical vein (Figure 8) can also become permeable and drain into the superior epigastric vessels and then into the system of the superior vena cava or through the inferior epigastric vessels to the external ilian vein and subsequently to the system of the inferior vena cava (5).

Hepatic pseudo-cirrhosis
There are pathologies that can alter the hepatic morphology and cause pseudocirrhosis, as in the case of patients with hepatic metastasis treated with chemotherapy. The treatment can create areas with scars where the tumoral tissue becomes retracted and areas of regenerative and healthy hepatic parenchyma. This condition is known as pseudocirrhosis due to its similar morphology with macronodular cirrhosis. Common findings in images include lobular margins, hypertrophy of segment 1 and signs of portal hypertension developed within a few weeks or months after treatment (Figure 9).

Figure 1. Micronodular cirrhosis.
CT with intravenous contrast: hepatic gland of irregular edges, which adopts a micronodular-type pattern (white arrows).

Figure 2. Macronodular cirrhosis.
Ultrasound (A) and axial CT with intravenous contrast (B) that shows a liver with irregular edges adopting a macronodular pattern in a patient with a history of chronic hepatitis B (arrows in A and B).
Figure 3. Fibrous surface.
CT with intravenous contrast in a late stage that shows enhanced fibrous bands in the hepatic parenchyma (black arrows). The hepatic gland presents nodular edges (white arrows) and an increase in the hilar periporal space. There is splenomegaly (asterisk) as a sign of portal hypertension.

Figure 4. Measurement of the hilar periporal space.
CT with intravenous contrast. Image A shows a normal hilar periporal space, smaller than 10 mm. Image B of a patient with alcoholic cirrhosis shows an increase of the hilar periporal space, which measures 15.9 mm. There is also hypertrophy of segment I (asterisk).

Figure 5. Atrophy of the medial segment.
CT with intravenous contrast showing atrophy of the medial hepatic segment in two patients with alcoholic cirrhosis (asterisk).
Figure 6. Posterior hepatic notch.
CT with intravenous contrast and T2-weighted MRI of two different patients with cirrhosis that shows a prominent groove in the right posterior surface of the liver (arrows in A and B).

Figure 7. Paraesophageal varices.
CT with intravenous contrast that shows large venous dilations (thick arrows) adjacent to the esophagus (thin arrow).

Figure 8. Repermeabilization of the paraumbilical vein.
CT with intravenous contrast that shows repermeabilization of the paraumbilical vein (arrows).
Figure 9. Hepatic pseudo-cirrhosis.
Female 65-year-old patient with a history of breast cancer presenting a liver with nodular edges (white arrows) and multiple hypodense space-occupying masses (black arrows), which correspond to secondary lesions. There is evidence of ascites (asterisks).

**Concusion**
Recognizing the hepatic morphological changes in images can help to diagnose cirrhosis in early stages. It is very important to determine the presence or absence of signs of cirrhosis to perform an adequate characterization of a focal hepatic lesion.

**Bibliography**