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MORPHOLOGICAL CHANGES IN LIVER PARENCHYMA IN LIVER CIRRHOSIS

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Annotation

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.

Keywords: hepar, hepatic cirrhosis, parenchyma, hepatic edge.

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. 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 or nodular and thick in cases of macronodular cirrhosis. 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. 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. 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.

All in all, 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.

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