Cirrhosis & Lesion Characterization at MR Imaging

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Cirrhosis is present in up to 90% of patients with hepatocellular carcinoma (HCC). Common causes of cirrhosis include *(a)* hepatitis C virus (55% of cases); *(b)* hepatitis B virus (16%); *(c)* alcohol consumption (13%); and *(d)* other causes, including cryptogenic nonalcoholic fatty liver disease and nonalcoholic steatohepatitis (16%). Nonalcoholic steatohepatitis is considered to be an advanced stage of nonalcoholic fatty liver disease. Viral hepatitis increases the risk of liver cancer some 20-fold. Other studies suggest that more than 75% of cases of liver cancer worldwide, and 85% of cases in developing countries, are caused by viral hepatitis. Exposure to aflatoxins is probably also an important factor in tropical areas, likely because of the suboptimal storage of certain food items such as peanuts.

Ongoing hepatocyte injury from viruses, alcohol consumption, and nonalcoholic fatty liver disease results in increased liver cell turnover, leading to the formation of regenerative nodules. The formation of regenerative nodules is an attempt by the liver to replace the damaged hepatocytes and compensate for lost liver function. Within regenerative nodules, some hepatocytes can undergo further gnomic changes with atypia and hence progress to liver cell dysplasia. With these changes, the nodules increase in size and cellularity, giving rise to the formation of dysplastic nodules and, finally, HCC. Hyperplasia is another important and relatively lesser-known aspect of HCC carcinogenesis.

Once a diagnosis of cirrhosis has been established, HCC develops at a rate of 1%–4% per year. In patients with cirrhosis caused specifically by hepatitis C virus, the risk of developing HCC is higher. Approximately 3.9 million persons in the United States are infected with hepatitis C virus. After 20 years, HCC will develop in 1.9%–6.7% of all patients with chronic hepatitis C virus infection. In contrast, 1–1.25 million persons are infected with hepatitis B virus, and the annual probability of HCC is 0.5% and 2.4% in hepatitis B virus–related chronic hepatitis and cirrhosis, respectively.

The overall 5-year survival rate for patients with HCC who undergo no treatment is less than 5%. HCC is less common in Western countries than in East or Southeast Asia, although its prevalence is rising. Age-adjusted HCC prevalence rates tripled between 1975 and 2005.

By understanding the transition from regenerative nodules through dysplastic nodules to HCC, one can more easily make sense of the complex nodularity often depicted in cirrhotic livers on imaging studies, particularly with multiple magnetic resonance (MR) pulse sequences.

MR imaging has emerged as an important imaging modality for assessing cirrhosis and its complications, such as HCC. For one thing, the introduction of faster sequences has allowed high-quality imaging of the entire liver with high intrinsic soft-tissue contrast. Second, the use of automated contrast detection methods in combination with faster sequences allows reproducible capture of the arterial phase, which is essential for the detection and characterization of HCC. Third, because of the lack of ionizing radiation, routine gadolinium-enhanced three-dimensional (3D) multiphasic imaging with high temporal and spatial resolution and fat suppression can be performed. Finally, MR imaging allows simultaneous evaluation of the background liver parenchyma and the liver lesions with use of a combination of sequences, including T2-weighted sequences, T1-weighted sequences (including chemical shift imaging), echoplanar diffusion-weighted sequences, dynamic gadolinium-enhanced 3D multiphasic imaging, and liver-specific delayed phase sequences (if contrast agents with hepatobiliary excretion are used).

In this regard, MR imaging is analogous to histopathologic analysis. The MR imaging sequences used for liver imaging can be regarded as different "stains" that provide specific items of information regarding the underlying disease processes. A combination of such findings facilitates understanding of the "big picture" and often elucidates the nature of the liver abnormalities.

In this presentation, I will describe the concept of stepwise carcinogenesis, as well as the histologic and gross pathologic features of liver nodules, including HCC. In addition, we discuss commonly used MR imaging sequences for liver imaging and offer a practical MR imaging–based approach for the evaluation of liver lesions in cirrhosis. We also discuss and illustrate the pertinent MR imaging findings and correlate these findings with the stepwise carcinogenesis of HCC.