Hepatocellular (HCC) is a common malignancy that frequently is associated with hepatitis and cirrhosis. Its incidence in the United States is rising, and has almost doubled over the past 20 years. This rise is caused, in part, by the epidemic of hepatitis C virus, which can lead to cirrhosis and HCC. Cirrhosis due to hepatitis C causes 70% of cases of HCC in Japan, and 30 to 50% in the United States. However, approximately 40% of HCC cases in North America occur in noncirrhotic livers. The relative risk of HCC in patients with cirrhosis due to hepatitis C is approximately 100 times the risk for patients with cirrhosis who are not infected, whereas patients with cirrhosis resulting from alcohol abuse or primary biliary cirrhosis have only a two- to fivefold increased risk of HCC. The median life expectancy of a patient with cirrhosis in whom HCC is untreated is only 13 months, with fewer than 33% of patients surviving 2 years.

Numerous treatment options are available, including surgical resection, alcohol or radiofrequency ablation, transcatheter chemoembolization, and liver transplantation. In the planning of treatment for patients with HCC successful long-term outcome is dependent on early detection of HCC, as well as accurate delineation of the number and location of tumor nodules. Early diagnosis of HCC and surgical resection or transplantation substantially increases survival. It is therefore critical to detect nodules that contain HCC at an early stage to control tumor burden while awaiting transplantation and to distinguish those patients who may have favorable long-term results with transplantation from those who will not.

The natural history of HCC suggests a stepwise carcinogenesis in patients with cirrhosis. According to this concept, a regenerative nodule increases in size and cellularity with changes of atypia and is transformed into a dysplastic nodule. A focus of HCC may develop within a dysplastic nodule. This focus can progress to a small HCC and then to a large (> 2 cm) HCC. Simultaneously, the nodule develops its own neovascularity, which is necessary to support the rapid growth of the nodule into HCC. The detection and characterization of hepatic nodular lesions on imaging depends on the contrast difference between the normal liver parenchyma and nodular lesions. The background of fibrosis can significantly affect the sensitivity of MRI in detection and characterization of these nodules.

HCC can present with different morphological subtypes including nodular, massive, and diffuse / infiltrative. Patients with nodular HCC can present with a single or multiple encapsulated nodules. Infiltrating HCC can be difficult to diagnose since it often lacks a well-demarcated boundary on cross-sectional imaging and can therefore
blend into the background of the cirrhotic liver. Many patients present with advanced disease and treatment can be challenging given, in part, to the difficulty in assessing the tumor extent within the liver. In addition, due to its large, diffuse nature and propensity to involve the portal vein, diffuse, infiltrating HCC may be difficult to treat and be associated with increased complications as well as marginal long-term benefit.

This presentation will discuss the spectrum of MR imaging findings during the stepwise carcinogenesis of HCC in cirrhosis. The value of different pulse sequences in making a diagnosis will be presented.

**Selected References**


