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- Precancerous & Cancer Mimicking Lesions in HBP Field -

Session 2. Mimickers of Hepatic Malignancy: What to Do?

Imaging findings of mimickers of hepatic malignancies

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Curriculum Vitae

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Imaging findings of mimickers of hepatic malignancies

With tremendous technical advances in liver imaging over the past few years, liver imaging plays central role in the diagnosis of hepatic tumor and the surveillance of high-risk populations for hepatocellular carcinoma (HCC). Liver MRI has much potential to improve both the detection and characterization of hepatic tumors as it provides multifactorial information using a variety of MR sequences—T1- and T2-weighted imaging, contrast-enhanced dynamic imaging and hepatobiliary phase imaging and diffusion-weighted imaging. HCC can be diagnosed radiologically, without the need for biopsy if the typical imaging features are present. Imaging criteria for the diagnosis of HCC that the European Association for the Study of the Liver (EASL) and the American Association for the Study of Liver Diseases (AASLD) employ are arterial hypervascularization and washout on portal venous and/or 3 min delayed phase on 4-phase CT and/or MRI. However, such typical vascular profile of HCC does not occur in considerable amount of HCC. Gadoxetic acid (Gd-EOB-DTPA), a hepatobiliary MR contrast agent for liver imaging, is widely used due to its excellent capability to delineate focal liver lesions including malignancy (HCC and metastasis) as hypointense on hepatobiliary phase image. By using gadoxetic acid, it is now easy to differentiate between arteriportal shunt (most common pseudolesion in cirrhotic background) and atypical arterial-only enhancing HCC without washout. In addition, by observing tumoral enhancement on hepatobiliary phase, we can easily characterize focal nodular hyperplasia mimicking hypervascular HCC as FNH is mostly seen as hyperhancing nodule with central scar area on hepatobiliary phase. Even though some HCC (~up to 10%) may show enhancement on hepatobiliary phase that is similar to FNH, the majority of FNH have area of central darker signal intensity than peripheral hyperintense rim on hepatobiliary phase, which is characteristic feature of FNH.

When only relying on enhancement pattern, overlapping among a variety of hepatic tumors quite exists. So, we need to discern many ancillary features specific for tumor. Internal heterogeneity is fundamental characteristic of HCC. This is attributed by expansive growth pattern of advanced HCC, resulting in capsule and intratumoral septum formation by condensation of fibrous elements of either surrounding noncancerous liver tissue (capsule) or the tumor tissue with the weaker growth activity compressed by the adjacent tumor tissue with more aggressive growth (septum). Capsule or septum is rarely seen in other hepatic tumors. Thus, we can benefit by applying capsule, intratumoral septum, and T2 bright foci representing multifocal hemorrhagic foci or peliosis to conventional enhancement feature for differentiating HCC from other hepatic tumors such as hepatocellular adenoma, angiomylipoma, and cholangiocarcinoma. Hepatic inflammatory conditions such as eosinophilic infiltration or small microabscess mimics HCC or metastasis. In particular, accurate differentiation of small hepatic abscess from metastasis is still challenging, particularly when it manifests as a small lesion in a patient who has underlying malignant biliary obstruction or underwent bile duct surgery, as both conditions are vulnerable to both microabscess and metastasis. Liver MRI that provides a variety of sequences including hepatobiliary phase makes it possible to differentiate such inflammatory con-
ditions from hepatic malignancy as most of inflammation tend to show size discrepancy between precontrast T1-weighted image (smaller or barely perceptible) and T2-weighted image, DWI, and hepatobiliary phase (showing clear signal change and larger than other sequences). Due to wide employment of gadoxetic acid, characterization of hemangioma is sometimes problematic because the gadoxetic acid begins to be taken up by hepatocytes approximately at 60 to 90 seconds after contrast injection and in turn hypervascular non-HCC tumor such as small cholangiocarcinoma and even hemangioma might be misdiagnosed as HCC because most of focal liver lesion tend to show hypointensity on portal venous or 3min late phase of gadoxetic acid MRI. In such case, we need to consider T2 brightness suggesting hemangioma. In cirrhotic background, hemangioma tends to show atypical imaging features such as internal sclerosis, which mimics atypical HCC or cholangiocarcinoma. Reactive lymphoid hyperplasia and bile duct adenoma are rare benign conditions but can be manifested as hypervascular mass mimicking HCC. No specific way has been advised for distinguishing such conditions from HCC. Therefore, clinicians should keep in mind that dissociation between imaging and pathology always exists even though imaging technology has evolved to a degree that HCC is not being diagnosed based on only imaging features without histologic confirmation.

References