Radiological Features of Hepatocellular Carcinoma

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Present article is a review of radiological features of hepatocellular carcinoma on various imaging modalities. With the advancement in imaging techniques, biopsy is rarely needed for diagnosis of hepatocellular carcinoma (HCC), unlike other malignancies. Imaging is useful not only for diagnosis but also for surveillance, therapy and assessing response to treatment. The classical and the atypical radiological features of HCC have been described. (J Clin Exp Hepatol 2014;4:S63–S66)

Present day radiology plays an essential role in evaluation and treatment together with surveillance of hepatocellular carcinoma. The detailed information regarding the various stages, lesion description, locoregional disease infiltration or distant disease spread can be ascertained. It is essential to use the imaging modalities deliberately and liberally to confidently detect early lesions; thereby allowing effective and curative treatment options for HCC.

CLASSIFICATION OF LIVER LESIONS

The liver lesions are classified into the following as per Liver Imaging Reporting and Data System (LI-RADS).[1]

1. Definitely benign (LR1)
2. Definitely HCC (LR5)
3. Probably HCC (LR4)
4. Probably benign (LR2)
5. Indeterminate (LR3) with equivocal imaging features

Smaller lesions (1–2 cm) must meet more stringent imaging criteria than larger lesions (2–5 cm) in order to be diagnosed as HCC on multiphase contrast enhanced imaging (CT or MRI). Lesions should have following features to meet criteria

Lesions between 1 and 2 cm must be hypervascular on arterial phase imaging, and demonstrate portal vein/delayed phase washout and pseudocapsule enhancement. If both washout and pseudocapsule enhancement are not present, they must demonstrate growth on serial imaging or confirmed on histology.[2,3]

Lesions between 2 and 5 cm or more must be hypervascular on arterial phase imaging and demonstrate portal vein/delayed phase washout or pseudocapsule enhancement. If no washout or pseudocapsule enhancement, lesion must demonstrate growth on serial imaging.

Lesions less than 1 cm are indeterminate (and thus, not eligible to be considered as HCC).

CHARACTERISTICS OF HEPATOCELLULAR CARCINOMA

Arterial Enhancement

Hepatic artery is the primary feeder to the HCC. It is important to optimize the protocol of imaging based upon the characteristic arterial phase enhancement of HCC. The avid contrast enhancement in arterial phase of the image acquisition is crucial to radiological diagnosis of HCC.[4]

With the evolution of dual source/dual energy and 32 channels MRI imaging, the detection of lesions smaller than even 1.0 cm is possible, especially on arterial phases. If the lesions are larger than 1.0 cm, than in addition to arterial phase imaging, a portal-delayed phase washout can be appreciated and is characteristic of HCC. From the initial three phase imaging (triple phase study) a recommendation of image acquisition for multiple sequences of the arterial phase is important (four-phase CT). This increases the sensitivity for smaller lesions with a profound neovascularization, circumventing the differences in blood flow kinetics and characteristics of the

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Abbreviations: A-P: arterio-portal; CT: computerized tomography; CTAP: CT during arterial portography; CTHA: computerized tomography hepatic angiography; Gd-BOPTA: gadopentetate dimeglumine; Gd-EOB: gadolinium ethoxybenzyl; Gd-EOB-DTPA: gadolinium ethoxybenzyl diethylenetriamine pentaacetic acid; GRE: gradient echo; HCC: hepatocellular carcinoma; LI-RADS: Liver Imaging Reporting and Data System; MDCT: multidetector CT; MRI: magnetic resonance imaging; SPIO: superparamagnetic iron oxide; T1W: T1 weighted; T2W: T2 weighted; TACE: trans-arterial chemo-embolization; TE: time to echo; THAD: transient hepatic attenuation differentiation; THID: transient hepatic intensity differentiation
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Imaging

tumor. Some large lesions may be hypovascular on arterial phase and may show a heterogenous delayed enhancement.

Washout

The term rapid washout is presence of hypodensity or hypointensity of the lesion as compared to rest of the liver on portovenous or delayed phases, and has a specificity of 95–96% for diagnosis of HCC.[4,5] Absence of a washout does not exclude HCC as some of these may appear hyperintense or isointense during portovenous phase.[6]

Capsule

About 90% of large HCC (>5 cm) in Asian countries and about 42% of cases in non-Asian countries, have a tumor capsule.[4] Presence of a capsule or a pseudocapsule differentiates HCC from regenerative and dysplastic nodules. On CT, the capsule usually is hypodense and on MRI hyperintense on T1W and T2W. Some studies suggested that, low-grade HCCs are well encapsulated and the efficacy of TACE treatment is better in HCCs with a capsule.

Vascular Invasion

Vascular invasion is common in large and/or high-grade tumors. Thrombosis of the portal vein, is seen 44–62.8% of large HCC. Thrombosis may be because of the tumor invasion or non-tumoral. The management strategy for the thrombus due to tumor and a bland thrombus are different and critical. Tumor thrombus is usually identified in contiguity of the primary tumor and tends to enlarge the adjacent vessels due to tumor encroachment.

The behavior of the tumor thrombus is identical to that of the parent tumor, exhibiting post contrast enhancement, and a high T2 intensity together with a washout and a restricted diffusion. There is a propensity for a higher ADC (apparent diffusion co-efficient) in the bland thrombus than HCC, more than the tumor thrombus itself. The large HCCs have a combination of areas of variable contrast enhancement and inherent signal intensity in T1 and T2 sequences which gives a mosaic appearance. On histopathology, there are multiple confluent areas of tumors, which have a variable cellular differentiation, adjacent parenchymal fibrosis, scarring, necrosis together with hemorrhage and septations, which explains the heterogeneous appearance.

Tumor thrombus	Non-tumor thrombus
Tumor in continuity with the vessel thrombosis	This may not be present
Expansile	Non-expansile
Tumor thrombus enhances post contrast, especially in arterial phase	No thrombus enhancement
Tends to have neovascularity	No neovascularity
Thrombosed vessel appears larger	Appears shrunken/normal size

IMAGING FEATURES OF VARIANTS OF HEPATOCELLULAR CARCINOMA

Hypervascular Nodules Without Washout

Based upon the nature of contrast uptake in the arterial phase, the liver lesions can be hypovascular and hypervascular in relation to the normal hepatic parenchyma. Sometimes, there is a delayed contrast enhancement, without any washout. Such lesions may be focal nodular hyperplasia, hemangiomas, hepatic adenoma, hypervascular metastases are of transient hepatic enhancement difference. Some HCC may not show washout with conventional imaging and would either need newer imaging modalities like Gd-EOB-DTPA MRI and/or biopsy for confirmation of diagnosis.[7]

Hypovascular Nodules

Hypovascular liver lesions are frequently identified and most likely metastases. About 10% of HCC are hypovascular. Delayed enhancement is found in malignant tumors like cholangiocarcinoma.

Diffuse Hepatocellular Carcinoma

Poorly demarcated, heterogeneously diffuse, infiltrating lesions representing HCCs are not uncommon. On MRI such lesions have a variable but homogenous T1-T2W appearances (T1 hypointensity and T2 hyperintensity).[8] Diffuse infiltrating HCCs demonstrate hypoenhancement or nodular patchy enhancement on arterial and heterogenous reticular enhancement on delayed contrast phases.

Portal vein thrombosis is common in such diffuse infiltrating HCC (5–44%).

Hepatocellular Carcinoma in Non-cirrhotic Liver

The overall reported prevalence is upto 54% of all HCC especially in oriental countries. Fibrolamellar HCCs are major differentials. HCCs in non-cirrhotic livers are larger, well-demarcated, solitary lesions with large areas of necrosis and are usually diagnosed at a later stage. Such HCCs uncommonly may show calcification, fibrosis and central scar.[1]

RADIOLOGICAL DIFFERENTIALS

Transient Hepatic Intensity Differentiation (THID)/Transient Hepatic Attenuation Differentiation (THAD)

THAD/THIDs are peripherally located, wedge shaped, segmental liver parenchymal areas formed due to portal flow redistribution in proximity to focal lesion, mostly