The imaging diagnosis of hepatocellular carcinoma is challenging as benign hypervascular lesions and arterioportal shunts (pseudolesions) often mimic it. There is also overlap in the imaging appearance from dysplastic and regenerating nodules. This article addresses the above imaging problems, examines proposed non-invasive imaging criteria for the diagnosis of hepatoma and discusses the optimal imaging modality.

**Keywords:** CT; MR; US; microbubble; hepatocellular carcinoma.

**Introduction**

Hepatoma, or hepatocellular carcinoma (HCC), is an epidemiologically significant tumor worldwide. It is the fifth most common tumor in the world\(^1\). It is particularly prevalent in Asia where hepatitis B infections are endemic. HCC has generated interest in Europe and the USA as its incidence has increased in the past decade. Several articles have reviewed the imaging features of HCC\(^2-8\). This review aims to provide a succinct summary of the issues confronting the radiologist in the assessment of HCC.

**Surveillance**

Ultrasonography (US) is a widely accepted imaging modality for HCC screening that is cheap, safe and repeatable. Due to its widespread adoption, it is difficult to recruit patients into a non-screening arm of a randomized trial. To date, there are no randomized controlled trials to validate the value of US in screening for HCC. In a large study employing both US and serum alpha fetoprotein (AFP) involving 1069 non-cirrhotic hepatitis B carriers, 14 HCC were detected. US had a sensitivity of 78.8% and specificity of 93.8%\(^9\). In general, HCC detection rates of surveillance are between 2 and 10% depending on the length of follow-up\(^10\). The mean doubling time of HCC on US is 180 days and hence 6 months is a reasonable screening time interval\(^11-13\).

A consensus statement from the European Association for the Study of the Liver (EASL) suggested surveillance based on AFP and US every 6 months\(^14\). It is recommended that only patients who would benefit from surgery be screened. Hence screening is suggested for non-cirrhotic patients and Child–Pugh’s A cirrhotic patients. Child–Pugh’s B cirrhotics would benefit from screening if transplantation was available. Child–Pugh’s C cirrhotics should be considered for transplantation and if this is not available, surveillance is pointless.

The positive predictive value of US is low (14%) and hence there is a need for recall and work-up protocols\(^15\). Such a strategy was proposed by the EASL\(^14\). The suggested work-up protocol relies on the size of the nodule detected. Nodules less than 1 cm are deemed too small to be accurately evaluated and three monthly US is suggested to assess nodule growth. For nodules larger than 2 cm in a patient with cirrhosis, a presumptive diagnosis of HCC is made based on non-invasive criteria elaborated in a later section. For cirrhotics with nodules between 1 and 2 cm, fine-needle aspiration cytology is
suggested. However, needle biopsy is not recommended if the lesion is surgically curable. Spiral or multidetector computed tomography (CT) is suggested to evaluate patients with elevated AFP but negative US examination.

**Imaging characteristics of HCC**

HCC is a hypervascular tumor with increased angio- genetic supply and hepatic arterial supply. It has a pseudocapsule composed of collagenous fibers and a layer of compressed liver tissue. Classically the tumor demonstrates hepatic arterial enhancement. During the portal venous and equilibrium phases, the tumor fades off while the pseudocapsule enhances brightly\(^7\). HCC often shows a mosaic pattern composed of several nodular components placed together like mosaic tiles. The mosaic pattern may be related to the multiclonal nature of the tumor\(^{16}\). Each nodular component can be thought of as arising from a clone of tumor cells with its own pseudocapsule. When two or more nodular components meet, their common boundary would form the internal septae, which would also enhance in the portal venous phase, just like the pseudocapsule (Fig. 1).

The above gross pathological features form the basis of imaging for HCC and are exploited by the various modalities. The pseudocapsule and internal septa are seen grossly in 80% of hepatomas\(^{17}\). They are more commonly seen in larger tumors (Fig. 2). On magnetic resonance (MR) scans, the pseudocapsule is seen in 67%, the internal septa in 43% and the mosaic appearance in...
Figure 2  HCC in a chronic hepatitis C carrier. (a) Arterial phase helical CT shows a multinodular mosaic pattern with a central necrotic scar. (b) Delayed phase helical CT shows an enhancing pseudocapsule (white arrow). (c) The cut surface of the gross specimen shows a similar multinodular mosaic pattern. Note the presence of the pseudocapsule (black arrows).

63%, whereas the pseudocapsule is seen in 31% and the mosaic pattern in 46% of CT examinations\cite{16,18}. The diagnosis of HCC is raised when these imaging features are seen in hypervascular lesions.

**Imaging diagnosis of hepatoma**

It is tempting to make a non-invasive imaging-based diagnosis of HCC, especially in the cirrhotic patient, as liver biopsies have attendant risks of hemorrhage and seeding (3%)\cite{19}. The EASL has proposed a set of non-invasive criteria for HCC in cirrhotic patients\cite{14}. The diagnosis is established if two imaging modalities (US, CT, magnetic resonance imaging (MRI)) show a coincidental nodule with arterial hypervascularization regardless of AFP levels, or if a single modality shows a lesion when the AFP levels are more than 400 ng/ml. Histologic diagnosis is required if the patient is non-cirrhotic or if the lesions are smaller than 2 cm.

It is important to recognize the limitations of the presumptive diagnosis of HCC made by imaging and AFP. The imaging characteristics of HCC are not pathognomonic. Differential diagnosis of hypervascular lesions includes hypervascular metastases, adenoma, FNH, angiomylipomas and Type 1 hemangioma\cite{7}. Small enhancing nodules in the hepatic arterial phase, even if they appear round or oval, may represent arterioportal shunts and pseudolesions\cite{20–24}. The signal characteristics of HCC on MRI show considerable overlap with dysplastic nodules and other lesions\cite{25}. Lipiodol uptake is also not pathognomonic as dense homogenous uptake can be seen in focal nodular hyperplasia (FNH) and patchy uptake in hemangioma, metastases, and FNH\cite{26}. AFP can be elevated in chronic hepatitis, fulminant hepatitis, cirrhosis and testicular tumor\cite{19,27,28}.

Hence if a lesion is operable for cure, the procedure establishing the diagnosis should be the curative liver resection\cite{14,19}. 
Assessment of disease extent

Tumor node metastasis (TNM) staging is generally considered important in classifying tumors into prognostic groups [29]. However, in HCC, patient survival is also dependent on the functional reserves of the liver, which is often cirrhotic. The Okuda classification takes into account both tumor burden and liver function (bilirubin, albumin, ascites) [30]. Although it identifies end-stage cases, it does not discriminate well between early and advanced cases. More recent proposals attempt to improve the prediction of outcomes of advanced cases but none are universally accepted [31].

Figure 3  Pseudolesion. This patient is a chronic hepatitis B carrier with previous resection of HCC. MRI was performed to evaluate rising AFP levels. A small enhancing lesion was noted in the arterial phase of the axial gradient-echo fat-suppressed T1-weighted series (black arrow). This was not seen in the portal venous phase (white arrow). Although the lesion was reported as indeterminate, surgery was performed in view of the elevated AFP. The lesion was not found during surgery. The affected area was nevertheless resected and histology showed benign liver tissue.

The predominant role of imaging in staging HCC is to evaluate the involvement of the venous structures and to detect small additional lesions that may preclude surgery. Identification of additional enhancing nodule in the hepatic arterial phase does not necessarily imply multiplicity. Pseudolesions and benign hypervascular lesions may mimic HCC [7,20–24]. There is also considerable overlap in the appearance of dysplastic and regenerating nodules, some of which may show hypervascularity in the arterial phase [32,33]. These difficulties and pitfalls are discussed in the following sections.

Hypervascular nodule—is it a real tumor?

Arteriportal shunts and pseudolesions are increasingly being recognized as mimics of HCC. More than a third of cirrhotics can demonstrate small enhancing nodules in the arterial phase which are the result of perfusion anomalies [22]. A large proportion of these pseudolesions are round or oval in shape [22,23] (Fig. 3). MR is a good modality to distinguish them from true lesions as the great majority of pseudolesions show no corresponding signal changes on T2-weighted scans [22,24]. This criterion is not absolute as small HCC less than 2 cm may not manifest signal changes on T2-weighted scans, and 8–26% of pseudolesions may show high signal on T2-weighted scans [24,34]. Some pseudolesions show a lack of uptake of superparamagnetic iron oxide (SPIO) and retention of Lipiodol [20,35]. In advanced cirrhosis requiring transplantation, two-thirds of non-arterial enhancing nodules more than 5 mm with no corresponding MR signal changes on other sequences are related to HCC [36].

Despite imperfections, MR remains, in our opinion, the best modality at distinguishing pseudolesions from true lesions though overlap patterns exist. Lesions presumed to be pseudolesions should be followed up to confirm the diagnosis.

Hypervascular nodule and cirrhosis—is it HCC?

The presence of multiple nodules in the cirrhotic liver is a common imaging problem as the surgeon would often need to know the nature of each nodule before deciding on surgery. Nodules in the cirrhotic liver may be regenerative, dysplastic or neoplastic. It is generally accepted that dysplastic nodules are premalignant for HCC. A theory of stepwise transformation of low-grade dysplastic nodules into high-grade dysplastic nodules and finally into HCC has been proposed and some supporting evidence is available [37–39].

Distinguishing the various nodules in a cirrhotic liver is difficult. One appreciates the problems better if one understands the blood supply of the various nodules. The blood supply of dysplastic nodules and HCC has been extensively investigated by CT hepatic arteriography (CTHA) and CT arterioportography (CTAP) [40]. As a nodule transforms, there is initially a decrease in hepatic arterial flow as the native hepatic arteries undergo degeneration. With increasing de-differentiation, there is a subsequent increase in angiogenesis and hepatic arterial supply. As such, there is a theoretical break-point where the decrease in normal supply is balanced by an increase in angiogenic supply. This break-point occurs in high-grade dysplastic nodules or well-differentiated HCC (Fig. 4). As current imaging modalities rely on detection of increased hepatic arterial supply, it is not difficult to see how well-differentiated HCC or high-grade dysplastic nodules can be misdiagnosed.
Figure 4 Schematic diagram illustrating the decrease in normal paired hepatic arterial supply and the increase in angiogenic arterial supply as a nodule becomes more dysplastic and neoplastic. (Adapted from Hayashi M, Matsui O, Ueda K et al. Correlation between the blood supply and grade of malignancy of hepatocellular nodules associated with liver cirrhosis: evaluation by CT during intraarterial injection of contrast medium. AJR Am J Roentgenol 1999; 172: 969.) RN, regenerating nodule; LG, low-grade dysplastic nodule; HG, high-grade dysplastic nodule; WD, well-differentiated HCC; MD, moderately differentiated HCC; PD, poorly differentiated HCC. Note that there is a point in the continuum where the decrease in normal hepatic arterial supply is balanced by an increase in angiogenic supply. This occurs in high-grade dysplastic nodules or well-differentiated HCC and explains the difficulty in diagnosis.

Explant studies have shown the poor sensitivity of CT, MR and angiography for small (less than 2 cm) HCC[41–43]. The overall sensitivity of MR is 55% and specificity 86% in the explanted liver.[43] The sensitivity of detecting HCC smaller than 1 cm is only 33%. There is also considerable overlap in the imaging patterns between HCC and dysplastic nodules. Differentiation from HCC on the basis of MR signal is difficult although HCC generally have a high signal on T2-weighted scans[25,44].

Freeny found that 41 (67%) out of 61 hypervascular nodules on CT in patients undergoing transplantation were related to benign regenerating nodules; three (5%) were dysplastic nodules and only 17 (28%) were HCC[32]. Lim found that hepatic arterial supply was increased in 21% of low-grade dysplastic nodules and hypervascularity was absent in 38% of high-grade dysplastic nodules[33]. Matsui found that 6% of HCC do not show increased hepatic arterial supply[45].

Notwithstanding these difficulties, it is still generally accepted that a nodule demonstrating increased arterial flow in a cirrhotic liver should be treated as a HCC. Nodules that do not show early enhancement are probably benign but should be followed up.

Which imaging modality is best?

The ideal imaging modality must be sensitive in detecting hypervascular lesions. It must be able to distinguish between arteriportal shunts and true lesions. It should be able to identify the supporting imaging features of HCC such as pseudocapsule, internal septa and mosaic appearance.

Dynamic contrast-enhanced MR is the optimal modality for the above reasons. Dynamic contrast-enhanced MR is more sensitive than dynamic contrast-enhanced CT[42,46,47] (Fig. 5). A recent explant study showed that MR detected more small lesions between 1 and 2 cm compared with CT (84 vs. 47%)[36]. Studying the impact on management, MR indicated the correct decision in 90% compared with 77–80% for CT[36]. CT remains a useful modality in view of its availability.

Conflicting data exist regarding the relative sensitivity of CTHA/CTAP and MR[48–51]. However, due to the problem of pseudolesions with CTHA and CTAP, MR is generally preferred. Jang found that combined CTHA and CTAP detected 20 more hypervascular lesions than dynamic CT in 52 patients[52]. However, only two of the 20 lesions were HCC while the remainder were proven to represent pseudolesions.

MR is useful in identifying pseudolesions as a great majority of them do not show corresponding signal changes on the unenhanced T1- and T2-weighted scans. However, small HCC can behave like pseudolesions and follow-up scans are required to confirm the absence of growth[22].

Angiography has poor sensitivity even compared to CT[53]. The use of Lipiodol-CT is not recommended by the EASL due to its limited accuracy[14].

New contrast agents

New US microbubble contrast agents show promise in the evaluation of HCC. Although there are limited data on the use of microbubble contrast agents in hepatoma, contrast-enhanced US can detect small HCC occult on MR[54]. Microbubble-enhanced US correlates well with CT in the assessment of response to radiofrequency (RF) ablative therapy[55–57]. There is tremendous potential for the use of microbubble contrast in the identification of HCC during RF ablation. It can potentially increase the sensitivity and accuracy of intraoperative US examinations and help characterize the ‘new’ lesion found in the operating room.

Liver-specific (hepatocyte and reticuloendothelial) MR contrasts face problems of the well-differentiated HCC behaving like normal liver parenchyma and demonstrating contrast uptake. They currently have a problem-solving role in certain situations[8].
Conclusion

Imaging diagnosis of HCC remains difficult. The diagnosis is suspected when a lesion demonstrates increased arterial flow, pseudocapsule, internal septa and a mosaic appearance. However, the radiologists should be aware of the pitfalls of pseudolesions and the overlap with dysplastic and regenerating nodules. MR appears to be a superior imaging modality although diagnosis of small (less than 2 cm) HCC remains difficult.

Acknowledgements

The authors would like to thank Dr Alexander Chung, Department of General Surgery, Singapore General Hospital for supplying the gross specimen photograph used in Fig. 2(c).

References

[1] Parkin DM, Bray F, Ferlay J, Pisani P. Estimating the world cancer burden: Globocan 2000. Int J Cancer 2001; 94: 153–6.
[2] Szklaruk J, Silverman PM, Charnsangavej C. Imaging in the diagnosis, staging, treatment, and surveillance of hepatocellular carcinoma. AJR Am J Roentgenol 2003; 180: 441–54.
[3] Frazer C. Imaging of hepatocellular carcinoma. J Gastroenterol Hepatol 1999; 14: 750–6.
[4] Kamel IR, Bluemke DA. Imaging evaluation of hepatocellular carcinoma. J Vasc Interv Radiol 2002; 13: S173–84.
[5] Murakami T, Kim T, Nakamura H. Hepatitis, cirrhosis, and hepatoma. J Magn Reson Imaging 1998; 8: 346–58.
[6] Ngan H. Imaging and radiological intervention in hepatocellular carcinoma. Hong Kong Med J 1997; 3: 57–68.
[7] Yu SC, Yeung DT, So NM. Imaging features of hepatocellular carcinoma. Clin Radiol 2004; 59: 145–56.
[8] Thng CH, Kuo Y, Blomley MJ. Imaging hepatocellular carcinoma. Cancer Rev Asia-Pacific 2003; 1: 191–210.
[9] Sherman M, Peltekian KM, Lee C. Screening for hepatocellular carcinoma in chronic carriers of hepatitis B virus: incidence and prevalence of hepatocellular carcinoma in a North American urban population. Hepatology 1995; 22: 432–8.
[10] Cottone M, Turni M, Caltagirone M et al. Screening for hepatocellular carcinoma in patients with Child’s A cirrhosis: an 8-year prospective study by ultrasound and alphafetoprotein. J Hepatol 1994; 21: 1029–34.
[11] Sheu JC, Sung JL, Chen DS et al. Growth rate of asymptomatic hepatocellular carcinoma and its clinical implications. Gastroenterology 1985; 89: 259–66.
[12] Ebara M, Ohto M, Shinagawa T et al. Natural history of minute hepatocellular carcinoma smaller than three centimeters complicating cirrhosis. A study in 22 patients. Gastroenterology 1986; 90: 289–98.
[13] Barbara L, Benzi G, Gaiani S et al. Natural history of small untreated hepatocellular carcinoma in cirrhosis: a multivariate analysis of prognostic factors of tumor growth rate and patient survival. Hepatology 1992; 16: 132–7.
[14] Bruix J, Sherman M, Llovet JM et al. Clinical management of hepatocellular carcinoma. Conclusions of the Barcelona-2000 EASL conference. European Association for the Study of the Liver. J Hepatol 2001; 35: 421–30.
[15] Collier J, Sherman M. Screening for hepatocellular carcinoma. Hepatology 1998; 27: 273–8.
[16] Honda H, Onitsuka H, Murakami J et al. Characteristic findings of hepatocellular carcinoma: an evaluation with comparative study of US, CT and MRI. Gastrointest Radiol 1992; 17: 245–9.
[17] Primary liver cancer in Japan. Clinicopathologic features and results of surgical treatment. Liver Cancer Study Group of Japan. Ann Surg 1990; 211: 277–87.
[18] Stevens WR, Johnson CD, Stephens DH, Batts KP. CT findings in hepatocellular carcinoma: correlation of tumor characteristics with causative factors, tumor size, and histologic tumor grade. Radiology 1994; 191: 531–7.
[19] Ryder SD. Guidelines for the diagnosis and treatment of hepatocellular carcinoma (HCC) in adults. Gut 2003; 52(Suppl 3): iii1–8.
[20] Kim TK, Choi BI, Han JK, Chung JW, Park JH, Han MC. Nontumorous arterioportal shunt mimicking hypervascular tumor in cirrhotic liver: two-phase spiral CT findings. Radiology 1998; 208: 597–603.
[21] Imai Y, Matsu O. Blood flow and liver imaging. Radiology 1997; 202: 306–14.
[22] Shimizu A, Ito K, Koike S, Fujita T, Shimizu K, Matsunaga N. Cirrhosis or chronic hepatitis: evaluation of small (< or =2-cm) early-enhancing hepatic lesions with serial contrast-enhanced dynamic MR imaging. Radiology 2003; 226: 550–5.
[23] Jeong YY, Mitchell DG, Kamishima T. Small (<20 mm) enhancing hepatic nodules seen on arterial phase MR imaging of the cirrhotic liver: clinical implications. AJR Am J Roentgenol 2002; 178: 1327–34.

[24] Kanematsu M, Kondo H, Semelka RC et al. Early-enhancing non-neoplastic lesions on gadolinium-enhanced MRI of the liver. Clin Radiol 2003; 58: 775–86.

[25] Earls JP, Theise ND, Weinreb JC et al. Dysplastic nodules and hepatocellular carcinoma: thin-section MR imaging of explanted cirrhotic livers with pathologic correlation. Radiology 1996; 201: 207–14.

[26] Nga H. Lipiodol computerized tomography: how sensitive and specific is the technique in the diagnosis of hepatocellular carcinoma? Br J Radiol 1990; 63: 771–5.

[27] Sherman M. Alpha fetoprotein: an obituary. J Hepatol 2001; 34: 603–5.

[28] Trevisani F, D’Intino PE, Morselli-Labate AM et al. Serum alpha-fetoprotein for diagnosis of hepatocellular carcinoma in patients with chronic liver disease: influence of HBSAg and anti-HCV status. J Hepatol 2001; 34: 570–5.

[29] Vauthey JN, Lauwers GY, Esaola NF et al. Simplified staging for hepatocellular carcinoma. J Clin Oncol 2002; 20: 1527–36.

[30] Okuda K, Ohtsuki T, Obata H et al. Natural history of hepatocellular carcinoma and prognosis in relation to treatment. Study of 850 patients. Cancer 1985; 56: 918–28.

[31] Llovet JM, Burroughs A, Bruix J. Hepatocellular carcinoma. Lancet 2003; 362: 1907–17.

[32] Freeny PC, Grossholz M, Kaakaji K, Schmiedl UP. Significance of hyperattenuating and contrast-enhancing hepatic nodules detected in the cirrhotic liver during arterial phase helical CT in pre-liver transplant patients: radiologic-histopathologic correlation of explanted livers. Abdom Imaging 2003; 28: 333–46.

[33] Lim JH, Cho JM, Kim EY, Park CK. Dysplastic nodules in liver cirrhosis: evaluation of hemodynamics with CT during arterial portography and CT hepatic arteriography. Radiology 2000; 214: 869–74.

[34] Yu JS, Kim KW, Jeong MG, Lee JT, Yoo HS. Nontumorous hepatic arterial-portal venous shunts: MR imaging findings. Radiology 2000; 217: 750–6.

[35] Mori K, Yoshioka H, Itai Y et al. Arterioportal shunts in cirrhotic patients: evaluation of the difference between tumorous and nontumorous arterioporal shunts on MR imaging with superparamagnetic iron oxide. AJR Am J Roentgenol 2000; 175: 1659–64.

[36] Burrel M, Llovet JM, Ayuso C et al. MRI angiography is superior to helical CT for detection of HCC prior to liver transplantation: an explant correlation. Hepatology 2003; 38: 1034–42.

[37] Anthony PP. Tumours and tumour-like lesions of the liver and biliary tract: aetiology, epidemiology and pathology. In: Pathology of the liver, 4th edition. MacSween RNM, Burt AD, ed. Edinburgh: Churchill Livingstone, 2002: 711–75.

[38] Lim AK, Patel N, Gdroyec WM, Blomley MJ, Hamilton G, Taylor-Robinson SD. Hepatocellular adenoma: diagnostic difficulties and novel imaging techniques. Br J Radiol 2002; 75: 693–9.

[39] Sakamoto M, Hirohashi S, Shimozato Y. Early stages of multistep hepatocarcinogenesis: adenomatos hyperplasia and early hepatocellular carcinoma. Hum Pathol 1991; 22: 172–8.

[40] Hayashi M, Matsu O, Ueda K et al. Correlation between the blood supply and grade of malignancy of hepatocellular nodules associated with liver cirrhosis: evaluation by CT during intraarterial injection of contrast medium. AJR Am J Roentgenol 1999; 172: 969–76.

[41] Krinsky GA, Nguyen MT, Lee VS et al. Dysplastic nodules and hepatocellular carcinoma: sensitivity of digital subtraction hepatic arteriography with whole liver exsplant correlation. J Comput Assist Tomogr 2000; 24: 628–34.

[42] Rode A, Bancel B, Douek P et al. Small nodule detection in cirrhotic livers: evaluation with US, spiral CT, and MRI and correlation with pathologic examination of explanted liver. J Comput Assist Tomogr 2001; 25: 327–36.

[43] Krinsky GA, Lee VS, Theise ND et al. Hepatocellular carcinoma and dysplastic nodules in patients with cirrhosis: prospective diagnosis with MR imaging and explantation correlation. Radiology 2001; 219: 445–54.

[44] Onaya H, Iai Y. MR imaging of hepatocellular carcinoma. Magn Reson Imaging Clin N Am 2000; 8: 757–68.

[45] Matsui O, Kadoya M, Kameyama T et al. Benign and malignant nodules in cirrhotic livers: distinction based on blood supply. Radiology 1991; 178: 493–7.

[46] Yamashita Y, Mitsuizuki K, Yi T et al. Small hepatocellular carcinoma in patients with chronic liver damage: comparative prospective analysis of detection with dynamic MR imaging and helical CT of the whole liver. Radiology 1996; 200: 79–84.

[47] Oh I, Murakami T, Kim T, Matsuhashi M, Kishimoto H, Nakamura H. Dynamic MR imaging and early-phase helical CT for detecting small intrahepatic metastases of hepatocellular carcinoma. AJR Am J Roentgenol 1996; 166: 369–74.

[48] Kanematsu M, Hoshi H, Murakami T et al. Detection of hepatocellular carcinoma in patients with cirrhosis: MR imaging versus angiographically assisted helical CT. AJR Am J Roentgenol 1997; 169: 1507–15.

[49] Murakami T, Kim T, Oi H et al. Detectability of hypervascular hepatocellular carcinoma by arterial phase images of MR and spiral CT. Acta Radiol 1995; 36: 372–6.

[50] Choi D, Kim SH, Lim JH et al. Detection of hepatocellular carcinoma: combined T2-weighted and dynamic gadolinium-enhanced MRI versus combined CT during arterial portography and CT hepatic arteriography. J Comput Assist Tomogr 2001; 25: 777–85.

[51] Kondo H, Kanematsu M, Hoshi H et al. Preoperative detection of malignant hepatic tumors: comparison of combined methods of MR imaging with combined methods of CT. AJR Am J Roentgenol 2000; 174: 947–54.

[52] Jang HJ, Lim JH, Lee SJ, Park CK, Park HS, Do YS. Hepatocellular carcinoma: are combined CT during arterial portography and CT hepatic arteriography in addition to triple-phase helical CT all necessary for preoperative evaluation? Radiology 2000; 215: 373–80.

[53] Takayasu K, Shima Y, Muramatsu Y et al. Angiography of small hepatocellular carcinomas: analysis of 105 resected tumors. AJR Am J Roentgenol 1986; 147: 525–9.

[54] Harvey CJ, Lim AK, Blomley MJ, Taylor-Robinson SD, Gdroyec WM. Cosgrove DO. Detection of an occult hepatocellular carcinoma using ultrasound with liver-specific microbubbles. Eur Radiol 2002; 12(Suppl 3): 70–3.

[55] Bartolozzi C, Lencioni R, Ricci P, Paolici A, Rossi P, Passariello R. Hepatocellular carcinoma treatment with percutaneous ethanol injection: evaluation with contrast-enhanced color Doppler US. Doppler Ultrasound 1998; 209: 387–93.

[56] Choi D, Lim HK, Kim SH et al. Hepatocellular carcinoma treated with percutaneous radio-frequency ablation: usefulness of power Doppler US with a microbubble contrast agent in evaluating therapeutic response—preliminary results. Radiology 2000; 217: 558–63.

[57] Wen YL, Kudo M, Zheng RQ et al. Radiofrequency ablation of hepatocellular carcinoma: therapeutic response using contrast-enhanced coded phase-inversion harmonic sonography. AJR Am J Roentgenol 2003; 181: 57–63.