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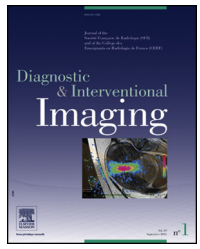

ORIGINAL ARTICLE /*Digestive*

MDCT features of hepatocellular carcinoma (HCC) in non-cirrhotic liver

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Abstract
Purpose: To describe the multidetector row computed tomography (MDCT) imaging features of HCC that develops in patients who are free from underlying liver cirrhosis and to determine if the MDCT presentation of this specific tumor differs from that of the more common HCC that develops in patients with liver cirrhosis using a retrospective case-control study.
Patients and methods: The MDCT examinations of 38 patients with HCC in non-cirrhotic liver (group 1) were quantitatively and qualitatively analyzed and compared to those obtained in 38 patients with HCC in cirrhotic liver (group 2) matched for age and gender. Quantitative and qualitative characteristics of HCC of both groups were compared using univariate analysis.
Results: HCCs were significantly larger in group 1 (81.5 mm ± 55.5) than in group 2 (44.5 mm ± 39.1 SD; $P = 0.0015$). In group 1, HCCs were more frequently single tumors (87%) than in group 2 (37%) ($P < 0.0001$), encapsulated (92% vs. 47% respectively; $P < 0.0001$), had more frequently fatty component (24% vs. 8%, respectively; $P = 0.0279$) and internal hemorrhage (29% vs. 3%, respectively; $P = 0.0033$). No significant differences were found between the two groups for location, hyperenhancement of HCC during the arterial phase, washout during the portal phase, endoluminal portal involvement by HCC, endoportal cruoric thrombus, invasion of adjacent organs and underlying liver steatosis.
Conclusion: HCC in non-cirrhotic liver are larger than those observed in cirrhotic liver and more frequently present as a single encapsulated tumor. They have the same patterns of enhancement than HCC that develops in cirrhotic liver.

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The imaging features of hepatocellular carcinoma (HCC) in patients with liver cirrhosis have been well described [1]. The main diagnostic criteria consist of hypervascularization during the arterial phase (i.e., the so-called ''wash-in'') and a subsequent washout during the portal phase of enhancement on contrast-enhanced imaging [2]. When these criteria are both present, the diagnosis of HCC is made with high degrees of confidence, thus obviating the need for further confirmatory tumor biopsy [3]. However, these criteria have been developed for HCCs occurring in patients with liver cirrhosis, so that they may not apply for HCCs that develop in patients with non-cirrhotic liver [4].

To our knowledge, a few studies have specifically addressed the issue of imaging presentation of HCC in patients without cirrhosis, although this specific tumor has distinctive features by comparison with the more common HCC that develops in liver cirrhosis [5,6]. Three studies have described imaging criteria for the diagnosis of HCC in non-cirrhotic liver [4,6,7]. Of these, two studies have stressed the importance of making the correct diagnosis of HCC occurring in non-cirrhotic liver because myriad other tumors may have similar presentation [4,6]. Indeed, hypervascular tumor arising in an otherwise healthy or non-cirrhotic liver may correspond to metastatic neuroendocrine tumor, angiosarcoma, epithelioid hemangioendothelioma, angiomyolipoma, focal nodular hyperplasia or hepatocellular adenoma [4].

The goal of this study was to describe the multidetector row computed tomography (MDCT) imaging features of HCC that develops in patients who are free from underlying liver cirrhosis and to determine if the MDCT presentation of this specific tumor differs from that of the more common HCC that develops in patients with liver cirrhosis using a retrospective case-control study.

Patients and methods

Patients

The database of our institution was queried from January 2003 to December 2014 inclusively to identify all patients with HCC that developed in non-cirrhotic liver who were investigated during this period. Initially retrieved patients were further included in the study when they had MDCT examinations available for review, a histopathologically confirmed HCC, normal liver function test results, no liver dysmorphia on MDCT and a METAVIR score $\leq$ F3. Exclusion criteria were the absence of MDCT examinations, absence of definite histological diagnosis of HCC and F4 METAVIR score. Our review board approved the retrospective data analysis. The need for informed consent was waived.

The final cohort study (group 1) consisted of 38 patients (35 men and 3 women) with a mean age of 68.5 years $\pm$ 11.5 SD (range, 32—83 years). Four patients had a prior history of viral hepatitis B (two chronic and two cured), and two patients had a prior history of viral hepatitis C. The alpha-fetoprotein level was available for 27/38 patients (71%). HCC was discovered incidentally in 27/38 patients (71%) or because of abdominal symptoms in 4/38 patients (11%), altered general status in 4/38, patients (11%), hemoperitoneum in 2/38 patients (5%) or during yearly follow-up for viral hepatitis in 1/38 patient (2%). Surgical resection was performed in 30/38 patients (79%).

The results of histopathological analysis were available for review in all patients. Tissue samples from HCC were obtained after surgical resection in 30/38 patients (79%) or after percutaneous biopsy in 8/38 patients (21%). All histopathological examinations were performed by a pathologist with a 15-year experience in hepatic tumors who established the Edmonson grade. Tissue samples were also obtained from the liver parenchyma to confirm the absence of cirrhosis. Fibrosis was classified according to METAVIR score. The METAVIR scoring system has been described elsewhere [8]; briefly, the METAVIR fibrosis score ranges from F0 to F4 (absent, minimal, moderate, severe and confirmed cirrhosis) [8].

A control group (group 2) was identified, which consisted of 38 patients who were matched for age and gender and who had undergone MDCT during the same period for HCC that developed in a cirrhotic liver. They were 38 patients (35 men and 3 women) with a mean age of 68.6 years $\pm$ 11 (SD) (range, 34—83 years). In this group, HCC was histopathologically confirmed in all patients, either after percutaneous biopsy in 36/38 patients (95%) or after surgical resection in 2/38 patients (5%). Cirrhosis was also histopathologically confirmed in all patients.

MDCT protocol

All patients underwent MDCT of the abdomen and pelvis using a VCT Lightspeed® (General Electric Healthcare, Milwaukee, USA) using a standardized multiphasic protocol that consisted of an unenhanced pass followed by three contrast-enhanced passes obtained at 10, 70 and 120 s after the start of intravenous administration of iodinated contrast material. A non-ionic iodinated contrast material (Iodixanol®, GE-Healthcare, Little Chalfont, UK) containing 350 mg of iodine per milliliter was injected at a dose of 1.5 mL/kg of body weight and administered at a rate of 3—4 mL/s using an automated power injector. Images were acquired in cephalocaudad direction, during one breath-hold while the patient was in supine position.

Imaging and reconstruction parameters were as follows: voltage, 120 kVp; tube current, 120—170 effective mA; axial resolution, 0.625 mm; beam collimation, 40 mm; gantry rotation time, 0.6 s; beam pitch, 0.6. After acquisition, CT data were reconstructed twice using a B30 soft tissue reconstruction kernel in the axial plane. A first set of axial images was obtained with a thickness of 2 mm at 2 mm intervals. A second set was obtained at 0.625 mm thickness at 0.5 mm intervals for multiplanar reconstructions and maximum intensity projection (MIP) views. In addition, all patients of both groups had MDCT of the thorax.

Image analysis

For this retrospective study, MDCT images were reviewed in a random manner using the workstation of a picture archiving system (PACS) by two experienced abdominal radiologists working in consensus. The two radiologists were aware of the original goal of the study but did not know if the patients had or not underlying liver cirrhosis. During the reading sessions, axial images, 3-mm thickness multiplanar reconstructions