CT Acquisition
Liver CT composed of unenhanced, hepatic arterial, portal venous, and equilibrium (delayed) phases was performed using 64-multiple detector CT scanners (Aquilion 64, Canon Medical Systems, Ottawara, Japan and LightSpeed VCT, GE Healthcare, Waukesha, WI, USA). Scanning parameters of liver CT were, 120 kVp, 189–200 mAs, 5-mm slice thickness with an overlap of 2.5 mm, table speed of 26.5–39.4 mm/rotation (pitch 0.83–1.07), and a single-breath-hold helical acquisition of 4–6 seconds, depending on liver size. 120 mL of nonionic contrast material (Ultravist 300, 300 mg/mL iopromide, Schering AG, Berlin, Germany) was injected intravenously through the antecubital vein using an automatic injector at a 3–4 mL/s rate. CT images were obtained in craniocaudal direction from dome of the diaphragm to the lower pole of the right kidney during a single breath-hold. Hepatic arterial phase scanning began 30–40 seconds after injection of contrast agent by means of a bolus-triggered technique (120 kVp; 40–60 mA; monitoring frequency from 12 seconds after the contrast injection, 1 second; trigger threshold, 100 HU in the descending aorta; delay from trigger to initiation of scan, 18 seconds). Portal and equilibrium phase scanning began 60–70 seconds and 180 seconds, respectively, after contrast injection. Coronal and sagittal images were also reconstructed with both a 3.0 mm slice thickness and reconstruction interval.

Magnetic Resonance Acquisition
All magnetic resonance (MR) images were acquired using a 3T whole-body MR system (Intera Achieva 3.0T, Philips Healthcare, Best, The Netherlands) with a 32-channel phased-array coil used as the receiver coil. MRI protocol included a respiratory triggered T1-weighted turbo field-echo in-phase and opposed sequence (repetition time [TR]/first echo time [TE], second TE, 10/2.3 [in-phase] and 3.45 [opposed-phase]; flip angle, 15°; matrix size, 256 x 194; bandwidth, 434.3 Hz/pixel), a respiration-triggered single-shot T2-weighted sequence with an acceleration factor of 2 or 4 (TR/TE, 1342/80; flip angle, 90°; matrix size, 320 x 256; and bandwidth, 506.4 Hz/pixel), a breath-hold multishot T2-weighted sequence with an acceleration factor of 2 or 4 (TR/TE, 2161/70; flip angle, 90°; matrix size, 400 x 280; and bandwidth, 235.2 Hz/pixel), with a 5 to 7 mm section thickness, a 1 to 2 mm intersection gap and a field of view (FOV) of 32–38 cm. For gadoxetic acid-enhanced MRI, unenhanced, arterial phase (20–35 s), portal phase (60 s), transitional phase (3 min) and 20 minutes delayed hepatobiliary phase (HBP) images were obtained using a T1-weighted three-dimensional turbo-field-echo sequence (T1 high-resolution isotropic volume examination: Thrive, Philips Healthcare) (TR/TE, 3.4/1.8; flip angle, 10°; matrix size, 336 x 206; and bandwidth, 995.7 Hz/pixel) with a section thickness of 2-mm and an FOV of 32–38 cm. MR fluoroscopic bolus detection technique was used for arterial phase imaging. The contrast agent was automatically administered intravenously at a rate of 1 mL/sec for a dose of 0.025 mmol/kg body weight using a power injector, followed by a 20-mL saline flush. A diffusion-weighted single-shot echo planar imaging with the simultaneous use of respiratory triggering was performed using a TR/TE of 1600/70; the TR was matched in each patient to the length of the respiratory cycle prior to gadoxetic acid-enhancement. The scanning parameters were as follows: a b-value of 0, 100, and 800 s/mm²; spectral pre-saturation with inversion recovery for fat suppression; matrix size, 100 x 100; acceleration factor of SENSE, 4.0; FOV, 35 x 35 cm; number of excitations, 4; slice thickness, 5 mm; slice gap, 1 mm; and 33 axial slices. Depending on the respiratory efficiency of each patient, the acquisition time for this sequence ranged from 3 to 4 minutes.

Imaging Factors
Tumor size was measured defined as maximum lesion diameter on HBP MR images. Tumor contour assessed on HBP MR images was categorized into irregular contour (non-nodular tumors with irregular margin that had budding portion at the periphery) or not (nodular tumors with smooth contour) (1). Typical hepatocellular carcinoma (HCC) enhancement pattern was when the lesion showed arterial hypervascularity and subsequent washout on portal or equilibrium phase with CT or on portal phase with MR images (2, 3). Tumor capsule was defined as a peripheral rim that shows smooth hyperenhancement in portal venous or equilibrium/transitional phase on CT or MR images (4). Intratumoral fat was determined when high signal intensity foci within a tumor on in-phase chemical shift imaging showed signal drop on opposed-phase (5). Satellite nodules were defined as tumors ≤2 cm in size and located ≤ 2 cm from the main tumor (6, 7). Arterial rim enhancement of
the tumor was defined as ring-like enhancement of the tumor with relatively hypovascular central areas in the arterial phase CT or MR images (7, 8). Peritumoral parenchymal enhancement was defined as crescent or polygonal shaped enhancement around the tumor on arterial phase, becoming iso- or subtle hypointense compared with background liver parenchyma on hepatobiliary MR images (9, 10). Pertumoral hypointensity on HBP was defined as wedge-shaped or flame-like hypointense hepatic parenchyma around the tumor on HBP (11). Nonhypervascular hepatobiliary hypointense nodules were defined as hypointense nodules on HBP of gadoxetic acid-enhanced MRI that are non-hypervascular on arterial phase (12, 13).

Pathologic Factors
Anatomic resection was defined as systematic removal of a hepatic segment confined by tumor-bearing portal tributaries and included hemihepatectomy, segmentectomy and subsegmentectomy based on Couinaud's classification. Nonanatomic resection was other type of resection such as wedge resection. Serosal invasion was defined as the invasion to the serosal of the liver, including the exposure from the liver surface, the tumor invasion of the adjacent organs, and tumor rupture (14). Safety margin was divided into more than 1 cm margin vs. 1 cm or smaller. Positive surgical margin was defined as the presence of tumor cells at the inked margin of resection. Satellite nodules on pathology refered to simultaneous occurrence of microscopic HCCs due to intrahepatic metastasis usually located within 2 cm from the primary tumor. Gross vascular invasion was when vessel invasion was not evident on MRI but was present on resected specimen. Microvascular invasion was as defined as a tumor within a peritumoral vascular space lined by endothelium that was visible only on microscopy.

REFERENCES