Keywords: intraductal papillary mucinous neoplasm, MDCT, pancreas, pancreatic neoplasm

DOI: 10.2214/AJR.04.1820

Received November 24, 2004; accepted after revision February 1, 2005.

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AJR 2006; 186:687–695
0361–803X/06/1863–687
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OBJECTIVE. The purpose of our study was to evaluate factors predictive of the presence of invasive carcinoma associated with intraductal papillary mucinous neoplasm (IPMN) of the pancreas on MDCT.

MATERIALS AND METHODS. Preoperative MDCT of 36 consecutive patients (23 men, 13 women; mean age, 66.6 years) who had undergone surgical resection and had a pathologic diagnosis of IPMN were retrospectively assessed. CT was performed with a 4-MDCT scanner with 120 mL of IV contrast material at an injection rate of 3 mL/sec. Arterial and venous phase images were acquired at 25 and 50–60 sec from the start of IV contrast administration. Type of ductal involvement, location, tumor size in branch duct type and combined type lesions, caliber of the main pancreatic duct, caliber of the common bile duct or common hepatic duct, and solid appearance of the lesion were assessed on CT and correlated with pathologic findings for invasive carcinoma.

RESULTS. Pathologic analysis revealed carcinoma in situ in seven patients (19%) and invasive carcinoma in 15 patients (42%) arising from the IPMN. With invasive carcinoma, the size of the tumor in branch duct type and combined type, and the caliber of the main pancreatic duct were significantly larger compared with the lesions without invasive carcinoma (4.7 ± 1.7 cm vs 2.6 ± 1.4 cm [p = 0.0007] and 9.3 ± 5.5 mm vs 4.6 ± 4.1 mm [p = 0.006], respectively). A solid mass (p < 0.001), dilatation of the common bile duct or common hepatic duct (≥ 15 mm), and the presence of a stent (p = 0.0004) were correlated with the presence of associated invasive carcinoma.

CONCLUSION. MDCT helped to predict invasive carcinoma associated with IPMN.

Intraductal papillary mucinous neoplasm (IPMN) is a distinct and now well-accepted clinical and pathologic entity. In 1986, Itai et al. [1] described radiologic findings of five mucinous cystic tumors of the pancreas associated with dilatation of the entire main pancreatic duct with or without dilatation of branch ducts on CT and sonography. In 1996, IPMN was defined by the World Health Organization as “an intraductal papillary mucin-producing neoplasm, arising in the main pancreatic duct or its major branches” [2]. It has been recognized and diagnosed with increasing frequency in recent years [3–5].

IPMN represents a spectrum of disease ranging from small benign adenomas through aggressive and lethal malignancy. IPMN has a relatively more favorable prognosis when compared with ductal adenocarcinoma of the pancreas [6] but is an important precursor to invasive adenocarcinoma of the pancreas. IPMNs with an associated invasive carcinoma have a significantly worse prognosis than IPMNs without an associated invasive carcinoma [7].

Pathologically, IPMNs can be divided into the three categories according to the criteria established by the World Health Organization [8]. These include IPMN adenoma, IPMN borderline, and intraductal papillary mucinous carcinoma. Intraductal papillary mucinous carcinoma can be further categorized into noninvasive lesions (carcinoma in situ) and invasive carcinomas [8]. The prevalence of malignant lesions associated with IPMN, including carcinoma in situ and invasive carcinoma, has been reported to be 31–88% [4, 7, 9–13].

The preoperative determination of the benign or malignant nature of an IPMN is important because it can guide the surgical approach and determine prognosis. Morphologic features predictive of malignancy reported in the literature include marked dilatation of the main pancreatic duct [6, 10, 11, 13–16], main duct...
type or combined type lesions [6, 17, 18], large mural nodules [11, 16], solid mass [13], diffuse or multifocal involvement [13], large tumors of the branch duct type [6, 11, 16], and a widely opened orifice of the papilla of Vater on endoscopic sonography and endoscopic retrograde pancreatography [14]. However, some of these factors are controversial. For example, prior studies showed large tumors with different threshold values of 3 cm [11, 16] to 6 cm [6] correlated with malignancy, although some studies did not show correlation between the size of the mass and the presence of malignancy [10, 18]. Marked dilatation of the main pancreatic duct has been reported to be associated with malignancy in many studies [6, 10, 11, 13–16] but was not confirmed in another study [18]. Limited data are available on MDCT regarding the assessment of malignancy in IPMN. The purpose of this study is to evaluate the CT findings of surgically resected IPMN of the pancreas and to assess predictive factors on MDCT for the presence of associated invasive carcinoma in IPMN.

Materials and Methods

Patients

From December 1999 to November 2002, 36 consecutive patients underwent preoperative 4-MDCT evaluation and surgical resection of the pancreas with a pathologic diagnosis of IPMN of the pancreas. This group consisted of 23 men and 13 women with an age range of 32–86 years (average age, 66.6 years). The interval between CT and surgery was 2–117 days (mean, 31.7 days). CT images of these patients were retrospectively assessed. This retrospective study was performed after study exemption from our institutional review board. Informed consent was not required.

Pathologic Diagnosis

Pathologically, IPMN was diagnosed in the resected specimen in all patients. The definition of an IPMN used in this study was a grossly visible, mucin-producing, predominantly papillary or, rarely, flat epithelial neoplasm arising from the main pancreatic duct or branch ducts, with varying degrees of ductal dilatation [19]. Pathologically, IPMNs were divided into four groups: adenoma, borderline, carcinoma in situ, and IPMN with invasive carcinoma. The classification was made on the basis of the World Health Organization definitions [8].

CT Technique

All MDCT examinations were performed on a Somatom Plus 4 Volume Zoom scanner (Siemens Medical Solutions) using detector collimation of 4 × 1 mm to obtain 1.25-mm slice thickness. The data were reconstructed at 1-mm intervals (0.25-mm overlap). The other parameters were 120 kVp, 140–180 mAs, and 0.5-sec rotation speed. After fasting for at least 2–3 hr, each patient ingested 750–1,000 mL of water over a 15–20-min period before scanning began. A scout topogram was obtained. Then, except in one case, arterial and venous phase images were acquired at 25 and 50–60 sec from the start of an IV contrast material injection. In one patient, one-phase scanning at 25 sec was obtained because the pancreatic lesion was incidentally found during evaluation of an aortic aneurysm. We injected 120 mL of iohexol (Omnipaque 350, Amersham Health) through the peripheral venous line at 3 mL/sec.

All image data were reconstructed with the body soft-tissue algorithm. All scanning data were then transferred to a free-standing workstation (Silicon Graphics O2), which runs 3D Virtuoso software (Siemens Medical Solutions), for subsequent review.

Image Analysis

Two radiologists independently reviewed the images from each CT examination on the workstation. They were aware of the diagnosis of IPMN but were unaware of the pathology results. The reviewers used source images and multiplanar reformatted and 3D display images. The workstation allowed the reviewers to edit CT volume data to create optimal reformatted and 3D images in real time at frame rates of 10–30 frames per second. CT display parameters, including width, level, opacity, and brightness, for the reformatted and 3D images were primarily chosen subjectively by each individual reviewer.

The following CT features were evaluated: First, the largest diameter of the main pancreatic duct. Second, the type of ductal involvement. The lesion was classified as branch duct type when a unilocular or multilocular cystic lesion was present with no associated main pancreatic duct dilatation greater than 3.0 mm, as main duct type when the largest diameter of the main pancreatic duct was greater than 3.0 mm with no associated unilocular or multilocular cystic lesion, and as combined type when both branch duct and main duct type features were present [6, 11]. Third, size. The size of the lesion obtained by the largest diameter of the lesion of the branch duct or combined type. The multilocular cystic lesion with septations was measured entirely, not the largest cystic component. The cystic and solid lesion was measured entirely, including the cystic and solid components, not only the cystic component. The size was not measured for main duct type lesions. Fourth, location. The location of the lesion was classified as head or uncinate, body, tail, or diffuse. When the main duct dilatation extended into more than one segment, or a branch duct type or combined type lesion involved more than one segment (head or uncinate, body, or tail), the lesion was classified as diffuse. Fifth, CT appearance of the branch duct type and combined type. The CT appearance was classified into three types: unilocular; multilocular cystic with thin septations or minimal mural nodule or minimal wall irregularity; and cystic and solid mass. The lesion was classified as a cystic and solid mass when a soft-tissue attenuation area was observed in the mural nodule or solid component in the cystic mass, or when a solid soft-tissue lesion around the cystic mass that showed a different degree of contrast enhancement from surrounding normal pancreatic parenchyma was present. For main duct type lesions, the CT appearance was not classified. Sixth, calcification within the lesion. Seventh, the largest diameter of the common bile duct or common hepatic duct. Dilatation of the common bile duct or common hepatic duct was considered present when the largest diameter of the common bile duct or common hepatic duct was equal to or greater than 15 mm or when a stent was present in the common bile duct. Eighth, other findings. Other findings, including vascular involvement, lymph node enlargement, duodenal involvement, and distant metastasis, were also recorded. When discrepancies were found between the findings of two radiologists, the CT examination was reviewed by the two radiologists and the final decision was made by consensus.

Statistical Evaluation

Correlation of the presence or absence of invasive carcinoma with each CT feature was performed. IPMN adenoma, IPMN borderline, and IPMN with carcinoma in situ were grouped together as noninvasive IPMNs for data analysis. Differences between categoric variables were evaluated using Fisher’s exact test. The unpaired Student’s t test was used to assess the correlation of continuous variables, including the diameter of the main pancreatic duct and the size of the lesion. A p value of less than 0.05 was considered significant. Interobserver agreement for the type of CT appearance was assessed using kappa statistics. Statistical analysis was performed with StatView-J 5.0 computer software (SAS Institute), Systat version 10.2 software (SYSTAT Software), and Stata version 8.0 software (Stata Corporation).

Results

Surgical Results

Total pancreatectomy was performed in four patients. Pancreatoduodenectomy was performed in 26 patients. Three patients underwent distal pancreatectomy and splenectomy, two patients underwent midsegment pancreatectomy, and one patient underwent pancreatectomy of the mid and distal segments.
**Pathologic Results**

All patients had a histologic diagnosis of IPMN. Among 36 patients, 15 patients (42%) had invasive carcinoma. Three patients had small or microscopic foci of invasive carcinoma. The average size of invasive carcinoma on the pathologic specimen in 12 patients, excluding the three patients with small or microscopic foci of invasive carcinoma, was 3.5 ± 1.3 cm. Seven patients (19%) had carcinoma in situ associated with IPMN. In 12 patients (33%), the lesion was classified as IPMN adenoma. In two patients (6%), the lesion was classified as IPMN adenoma. In one patient, IPMN borderline adenocarcinoma was found in the tail but there was also a separate invasive adenocarcinoma in the head that was not associated with IPMN. For statistical analysis, this patient was classified as IPMN borderline without invasive carcinoma. Among 15 patients with invasive carcinoma, six patients had duodenal involvement, eight patients had invasion into the common bile duct, and 10 patients had regional lymph node metastasis by pathologic examination, including two patients with direct extension of invasive carcinoma into the regional lymph nodes.

**Radiologic Findings**

**Largest diameter of the main pancreatic duct**—The average diameter of the main pancreatic duct in all patients was 6.5 ± 5.2 mm. The largest diameter of the main pancreatic duct in patients with invasive carcinoma was 9.3 ± 5.5 mm, and in patients without invasive carcinoma was 4.6 ± 4.1 mm. The diameter of the main pancreatic duct was significantly larger in patients with invasive carcinoma (*p* = 0.006) (Table 1).

**Type of ductal involvement**—Eleven patients (31%) were classified as branch duct type, 20 patients (56%) as combined type, and five patients (14%) as main duct type on the basis of CT findings. Among five main duct type lesions, two had diffuse involvement of the main pancreatic duct (Fig. 1) and three had segmental involvement of the main pancreatic duct (Fig. 2). Invasive carcinoma was present in two (40%) of the five patients with main duct type IPMN (Fig. 1), in one (9%) of the 11 patients with branch duct type IPMN (Fig. 3), and in 12 (60%) of the 20 patients with combined type IPMN (Figs. 4 and 5). By Fisher’s exact test analysis, the *p* value was 0.02, and a relationship was seen between the type of ductal involvement and the presence of invasive carcinoma (Table 2).

**Lesion size**—The average maximal diameter of the mass for the branch duct type and the combined type on CT was 3.5 ± 1.8 cm (range, 1.0–7.0 cm). The average size of the lesion in patients with invasive carcinoma was 4.7 ± 1.7 cm (Figs. 3–5), whereas the average size of the lesions in patients without invasive carcinoma was 2.6 ± 1.4 cm (Figs. 6 and 7). The average size of the lesion was significantly larger for both branch duct type and combined type IPMNs in patients with invasive carcinoma (*p* = 0.0007) (Table 1).

**Location of the IPMNs**—The most common location was head or uncinate, which was found in 22 patients (61%). Invasive carcinoma was present in 10 (45%) of these 22 patients. Five patients (14%) had a lesion in the body and three patients (8%) had a lesion in the tail of the pancreas. None of the patients with a lesion in the body or tail had invasive carcinoma. Among six patients who had diffuse involvement, five (60%) had associated invasive carcinoma. By Fisher’s exact test analysis, the *p* value was 0.01, and a relationship existed between the location of the lesion and the presence of invasive carcinoma (Table 2).

**CT appearance**—Among 31 branch duct type and combined type lesions, eight lesions (26%) were classified as unilocular on CT; seven (23%) as a multilocular lesion with thin septations, minimal mural nodule, or minimal wall irregularity (Fig. 6); and 16 (52%) as a cystic and solid mass (Figs. 3–5 and 7). For assessment of the type of CT appearance, the agreement between the two reviewers was poor (*κ* = 0.398). The disagreement was discussed by the two reviewers and a final decision was made. Invasive carcinoma was found in 12 lesions classified as cystic and solid and in only one lesion classified as cystic with thin septations, minimal mural nodule, or minimal wall irregularity. None of eight lesions classified as a unilocular lesion had invasive carcinoma. By Fisher’s exact test analysis, the *p* value was less than 0.001, a significant relationship existed between the CT appearance of the lesion and the presence of invasive carcinoma, and IPMN with invasive carcinoma tended to have a more solid appearance (Table 2).

**Calcification in the lesion**—Calcification in the lesion was seen in four patients, and invasive carcinoma was found in two of these four patients. No significant association was seen between the presence of calcification and invasive carcinoma (*p* > 0.99) (Table 1).

**Dilatation of the common bile duct**—Dilatation of the common bile duct equal to or greater than 15 mm or presence of common bile duct stent for obstructive jaundice—Five patients had a stent in the common bile duct that had been placed to relieve biliary obstruction, and four of these patients had invasive carcinoma arising in an IPMN in the head of the pancreas. One patient who had a stent in the common bile duct had an IPMN borderline lesion in the tail and also had infiltrating, moderately differentiated adenocarcinoma in the pancreatic head separate from the IPMN. The average of the largest diameter of the common bile duct or common hepatic duct was 11.3 ± 6.2 mm. The other 20 patients had no invasive carcinoma, and the average of the largest diameter of the common bile duct or common hepatic duct was 8.4 ± 4.9 mm. Among these 31 patients, 11 patients had invasive carcinoma associated with IPMN, and the average of the largest diameter of the common bile duct or common hepatic duct was 6.7 ± 3.2 mm (*p* = 0.096). Five patients had dilatation of the common bile duct equal to or greater than 15 mm on CT (Fig. 5). Among 10 patients with dilatation of the common bile duct equal to 15

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**TABLE 1: Average Main Duct Diameter and Maximal Size of Lesion in Branch Duct Type and Combined Type Without and With Invasive Carcinoma**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Without Invasive Carcinoma</th>
<th>With Invasive Carcinoma</th>
<th><em>p</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Main duct diameter (mm)</td>
<td>4.6 ± 4.1</td>
<td>9.3 ± 5.5</td>
<td>0.006</td>
</tr>
<tr>
<td>Maximal size (cm)</td>
<td>2.6 ± 1.4</td>
<td>4.7 ± 1.7</td>
<td>0.0007</td>
</tr>
<tr>
<td>Calcification</td>
<td>2</td>
<td>2</td>
<td>&gt; 0.99</td>
</tr>
<tr>
<td>Yes</td>
<td>19</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>9</td>
<td>0.0004</td>
</tr>
<tr>
<td>CBD or CHD dilatation (≥ 15 mm) or CBD stent</td>
<td>Yes</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Note—Data in last four rows are numbers of patients. CBD = common bile duct, CHD = common hepatic duct.</td>
<td>No</td>
<td>20</td>
<td>6</td>
</tr>
</tbody>
</table>
mm or more or a stent in the common bile duct, nine had associated invasive carcinoma ($p = 0.0004$) (Table 1). Infiltration of the invasive carcinoma into the distal common bile duct was pathologically confirmed in eight of these nine patients. Other findings—Lymph node enlargement was found in four patients on CT, but three were false-positive by pathologic examina-
tion. Pathologically, 10 (67%) of 15 patients with invasive carcinoma had metastatic carcinoma in the regional lymph nodes, including two patients with direct extension of invasive carcinoma to the regional lymph nodes. Duodenal involvement of the tumor was suspected in one patient, but six patients had duodenal involvement by pathologic examination. No vascular invasion to the celiac artery, superior mesenteric artery, main portal vein, splenic vein, or superior mesenteric vein was found on CT. Distant metastasis was not found on CT, and at surgery no patient was found to have distant metastasis.

**Discussion**

IPMN is defined as an intraductal papillary mucin-producing neoplasm arising in the main pancreatic duct or its major branch ducts. The papillary epithelial component and the degree of mucin secretion, cystic dilatation, and invasiveness are variable, and the
Fig. 5—Patient with invasive carcinoma arising in intraductal papillary mucinous neoplasm (IPMN). A and B, Axial (A) and coronal (B) reformatted venous phase images show 6-cm cystic mass in head of pancreas (arrows). Mild dilatation (< 10 mm) of main pancreatic duct is not shown. Common bile duct (arrowhead, B) is dilated (16 mm). Lesion was classified as combined type, cystic and solid lesion. Pathologically, invasive carcinoma measuring 5.5 cm in greatest dimension was seen arising in IPMN.

TABLE 2: Type of Duct Involvement, Location, and CT Appearance of Lesion with Associated Invasive Carcinoma

<table>
<thead>
<tr>
<th>Feature</th>
<th>No. of Patients</th>
<th>Without Invasive Carcinoma</th>
<th>With Invasive Carcinoma</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of duct involvement</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Branch duct</td>
<td>10</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>8</td>
<td>12</td>
<td></td>
<td>0.02</td>
</tr>
<tr>
<td>Main duct</td>
<td>3</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head or uncinate</td>
<td>12</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tail</td>
<td>3</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffuse</td>
<td>1</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CT appearance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilocular</td>
<td>8</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cystic with thin septations, minimal mural nodule, or minimal wall irregularity</td>
<td>6</td>
<td>1</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Cystic and solid</td>
<td>4</td>
<td>12</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Macrosopic appearance of IPMN and consequent imaging findings of the tumor vary according to the site of origin and the extent of ductal involvement of the disease [5].

IPMN represents a spectrum of disease from small benign adenomas through aggressive, lethal, and invasive malignancies. In the World Health Organization classification system [8], IPMNs are divided into categories depending on the degree of cytologic and architectural atypia. These categories include benign, borderline, and malignant noninvasive and invasive lesions. IPMNs with moderate dysplasia are placed in the borderline category. IPMNs with severe dysplastic epithelial change (carcinoma in situ) are designated as carcinoma even in the absence of invasion, and they can be papillary or micro-papillary. Cribriform growth and budding of small clusters of epithelial cells into the lumen support the diagnosis of carcinoma in situ [8]. Approximately half of the invasive carcinomas associated with IPMN are colloid (mucinous noncystic) carcinomas, and most of the remainder are ordinary tubular adenocarcinomas [19–21]. IPMNs with an associated colloid type of invasive carcinoma have a better prognosis than do IPMNs with an associated tubular type of invasive carcinoma [20]. Carcinoma in situ has been found in 7–42% of IPMNs, and invasive carcinoma in 25–46% [4, 7, 10, 12, 13]. In our study, carcinoma in situ was found in 19% and invasive carcinoma in 42% of patients.

Surgical resection is generally considered the treatment of choice for most IPMNs, and IPMNs resected before the development of invasive carcinoma are highly curable [3]. Preoperative detection of the presence or absence of associated invasive carcinoma is crucial in this disease. When invasive carcinoma is present, the surgical procedure is modified, with resection of regional lymph nodes. A poor outcome after surgery for patients with invasive carcinoma has been reported [7], particularly with node-positive invasive carcinoma associated with IPMN [3]. Some authors consider follow-up of patients instead of surgery for a branch duct type lesion when there is no invasion, when
MDCT of Pancreatic Neoplasms

Fig. 6—Patient with intraductal papillary mucinous neoplasm (IPMN) adenoma. A and B, Axial (A) and coronal (B) reformatted venous phase images show small cystic lesion in body of pancreas (arrows). Main pancreatic duct is visualized but not dilated (arrowheads, A). Lesion was classified as branch duct type, cystic with thin septations. Pathologically, lesion was IPMN adenoma.

Fig. 7—Patient with intraductal papillary mucinous neoplasm (IPMN) borderline lesion. A and B, Axial (A) and coronal (B) reformatted venous phase images show cystic lesion in head and uncinate process (3.3 cm) (white arrows). Note mild dilatation (4 mm) of main pancreatic duct (arrowhead, A). Pancreatic stent (black arrow, A) is present. Lesion was classified as combined type, solid and cystic lesion. Pathologically, lesion was IPMN borderline lesion.

the patient refuses surgery, or when the patient is in poor condition with other severe diseases [6].

Reported clinical predictive signs of malignancy in IPMN include diabetes mellitus [13, 15], history of alcohol abuse [22], short duration of symptoms [4], jaundice [4, 16, 19], elevated liver function tests [4], and elevated serum CA19-9 [4]. However, some of these
TABLE 3: Sensitivity and Specificity of CT Findings in the Diagnosis of Invasive Carcinoma Associated with Intraductal Papillary Mucinous Neoplasm of the Pancreas

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MPD involvement</td>
<td>92 (11/15)</td>
<td>48 (10/21)</td>
</tr>
<tr>
<td>Solid mass</td>
<td>92 (12/13)</td>
<td>76 (14/18)</td>
</tr>
<tr>
<td>MPD ≥ 6 mm</td>
<td>73 (11/15)</td>
<td>81 (17/21)</td>
</tr>
<tr>
<td>CBD or CHD ≥ 15 mm or CBD stent</td>
<td>60 (9/15)</td>
<td>95 (20/21)</td>
</tr>
<tr>
<td>CBD or CHD ≥ 10 mm or CBD stent</td>
<td>60 (9/15)</td>
<td>76 (16/21)</td>
</tr>
<tr>
<td>Tumor size ≥ 5 cm in branch duct type or combined type</td>
<td>54 (7/13)</td>
<td>94 (17/18)</td>
</tr>
<tr>
<td>CBD or CHD ≥ 20 mm or CBD stent</td>
<td>33 (5/15)</td>
<td>95 (20/21)</td>
</tr>
<tr>
<td>MPD ≥ 10 mm</td>
<td>33 (5/15)</td>
<td>86 (18/21)</td>
</tr>
<tr>
<td>MPD ≥ 15 mm</td>
<td>20 (3/15)</td>
<td>95 (20/21)</td>
</tr>
</tbody>
</table>

Note—Numbers in parentheses are numbers of cases. CBD = common bile duct, CHD = common hepatic duct, MPD = main pancreatic duct.

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Factors of invasive carcinoma of 69%, 83%, and 76%, respectively, using nonhelical CT. Using helical CT with unenhanced and two-phase contrast-enhanced protocols, Taouli et al. [13] reported sensitivity, specificity, and accuracy in the diagnosis of invasive carcinoma in IPMN of 96%, 89%, and 67%, respectively. Highly specific predictive signs of invasive carcinoma on CT reported by Taouli et al. included solid mass (sensitivity, 67%; specificity, 96%), main pancreatic duct greater than 10 mm (sensitivity, 78%; specificity, 92%), diffuse or multifocal involvement (sensitivity, 56%; specificity, 77%), and attenuating or calcified intraluminal content (sensitivity, 33%; specificity, 77%).

In our study, the CT features most specific for an invasive carcinoma associated with IPMN were common bile duct dilatation of 15 mm or more or the presence of a stent in the common bile duct for jaundice (sensitivity, 60%; specificity, 95%), main pancreatic duct dilatation of 15 mm or more (sensitivity, 20%; specificity, 95%), and tumor diameter of 5 cm or more in branch duct or combined type (sensitivity, 54%; specificity, 94%). A markedly dilated main pancreatic duct was associated with invasive carcinoma, and the sensitivity and specificity of CT in the diagnosis of associated invasive carcinoma vary depending on threshold value. When a threshold value of 15 mm was used, sensitivity and specificity were 20% and 95%, respectively. When a threshold value of 10 mm was used, they were 33% and 86%. When a threshold value of 6 mm was used, they were 73% and 81%. Highly sensitive signs in the diagnosis of invasive carcinoma were main pancreatic duct involvement (main duct type and combined type) (sensitivity, 93%; specificity, 48%) and solid mass (sensitivity, 92%; specificity, 78%) (Table 3).
Second, the type of ductal involvement and the location of the lesion were determined on the basis of CT findings and were not correlated with pathology findings. Taouli et al. [13] reported that CT and histopathologic correlation of the type of ductal involvement is poor. In their study, main pancreatic duct involvement was overestimated in two patients and underestimated in three patients, and false-negative cases of branch duct involvement occurred in two of 36 IPMN patients. Those authors also reported that the extent of IPMN was underestimated in two patients on CT [13]. They suggested that discrepancies of ductal involvement between CT and histologic findings might be explained as dilatation of the pancreatic duct due to associated chronic pancreatitis or obstruction by tumor growth or by mucous plugging produced by a tumor [13].

Third, we evaluated predictive factors for invasive carcinoma only but not for carcinoma in situ. On CT, carcinomas in situ and small invasive carcinomas remain difficult to detect. Fukukura et al. [10] reported that invasion into the pancreatic parenchyma was not depicted on thin-section helical CT in any of nine patients with malignant IPMN, including carcinoma in situ and invasive carcinoma, because they all had chronic pancreatitis and the invasion was minimal. In our study, a relatively large number of patients had macroscopic invasive carcinoma. Among 15 patients with invasive carcinoma, only three had small or microscopic foci of invasive carcinoma. The average size of invasive carcinoma, excluding these three patients, measured at pathologic examination was 3.5 ± 1.3 cm.

Fourth, we did not obtain unenhanced scans. Therefore, differentiating mural nodule or solid mass from mucin globes was occasionally difficult. Small intraductal areas of solid attenuation on CT can be caused by either intraductal mural nodules or mucin deposits and may be difficult to differentiate [1, 21, 23]. The mural nodule closely attaches to the wall and can be seen in the non-gravity-dependent position [1, 23], and it shows discrete enhancement [24]. Mucin glob can modify their position with variations in the gravity-dependent portion of the duct or isolated from the wall [23].

Fifth, the mean interval between CT and surgery was 31.7 days, and the disease may have progressed during that interval; however, this is probably of no significance in data analysis.

In conclusion, MDCT helped to predict invasive carcinoma associated with IPMN. Highly sensitive signs for the presence of invasive carcinoma in IPMN included main pancreatic duct involvement and a solid mass. Highly specific signs for invasive carcinoma were dilatation of the common bile duct or common hepatic duct (≥15 mm) or the presence of a stent in the common bile duct for obstructive jaundice, marked main pancreatic duct dilatation (≥15 mm), and a tumor of 5 cm or greater in branch duct type and combined type lesions on CT.

References