Intraductal papillary mucinous tumor (IPMT) of the pancreas was identified and classified only recently. IPMT has a primarily intraductal, papillomatous growth pattern, which is associated with excessive mucin secretion and results in progressive ductal dilatation or cyst formation. The tumor occurs in four forms: segmental or diffuse involvement of the main pancreatic duct and macrocystic or microcystic involvement of a branch duct. In the past, many IPMTs may have been misdiagnosed as chronic pancreatitis because of their generally benign behavior. The correct diagnosis, once achieved only with endoscopic retrograde cholangiopancreatography (ERCP), can now be made with noninvasive imaging modalities, particularly computed tomography (CT) and magnetic resonance (MR) imaging. ERCP remains the imaging modality of choice for diagnosis of IPMT. With ERCP, the communication between the cystically dilated ductal segment or branch duct and the main pancreatic duct is easily demonstrated. However, reflux of contrast material due to an excess of mucin or an enlarged papillary orifice hinders filling of the ductal tree. Filling defects due to mucin globs or mural nodules are also important clues to the diagnosis. Bulging of the papilla into the duodenal lumen is virtually pathognomonic of IPMT and is well demonstrated with CT or MR imaging.

Abbreviations: ERCP = endoscopic retrograde cholangiopancreatography, IPMT = intraductal papillary mucinous tumor, MPD = main pancreatic duct

Index terms: Pancreas, CT, 77.1211 • Pancreas, cysts, 77.3125 • Pancreas, MR, 77.1214 • Pancreas, neoplasms, 77.3125 • Pancreatic ducts, 774.3125

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**INTRODUCTION**

When cystic tumors of the pancreas are encountered in clinical practice, their detection raises problems with clinical decision making. The morphologic and structural features of cystic pancreatic masses (eg, serous cystadenomas, mucinous cystic tumors, solid and papillary epithelial neoplasms, cystic islet cell tumors) are well described (1–3).

Mucinous pancreatic tumors have been subdivided into peripheral (cystadenoma, cystadenocarcinoma) and ductal tumors according to their site of origin (4). The biologic behavior of these tumors is variable (Table); different histologic patterns frequently coexist in the same tumor (5,6). Ductal tumors originate from the main pancreatic duct (MPD) or its branches (4) and demonstrate four main patterns at imaging (Fig 1).

Intraductal tumors have been referred to by different names (mucinous ductal ectasia, papillary adenocarcinoma, ductectatic tumor, intraductal mucin-hypersecreting neoplasm, mucinous villous adenomatosis) (4); however, in 1997, the unified term *intraductal papillary mucinous tumor* was adopted (5). This term is based on the common origin of the lesion from the epithelial lining at any level within the pancreatic ductal system. IPMT has a primarily intraductal, papillomatous growth pattern, which is associated with excessive mucin secretion and results in progressive ductal dilatation or cyst formation.

The recent “discovery” of this lesion is due to the fact that many patients affected by this clinical entity in the past were thought to have chronic pancreatitis. Moreover, the benign behavior of many IPMTs justifies a clinical approach similar to that for pancreatitis. In 1982, Ohhashi et al (7) reported the endoscopic appearance of mucinous tumors. Later, the same aspects were demonstrated by other investigators, who defined the category of benign intraductal mucin-producing pancreatic tumors (4). The gross appearance of these tumors depends on their site of origin. Itai et al (8) described the radiologic aspects of branch duct–type tumors in 1986.

In this article, multiple aspects of main duct IPMT and branch duct IPMT are presented. Useful criteria for characterizing the lesion and assessing the grade of malignancy preoperatively are discussed. Imaging of IPMT is extremely important not only to identify the tumor but also to suggest the most appropriate therapeutic strategy in relation to the site of origin and size of the lesion.

**MAIN DUCT IPMT**

IPMT of the MPD is difficult to recognize because many of the radiologic features mimic the main duct dilatation seen in chronic pancreatitis. The involvement of the MPD may be segmental or diffuse (Fig 1a, 1b). Main duct IPMT occurs with equal frequency in male patients and female patients. For both sexes, the peak

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**Table: Mucinous Cystic Tumors of the Pancreas**

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<td><strong>Benign</strong></td>
<td>Mucinous cystadenoma</td>
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<td>Intraductal papillary mucinous adenoma</td>
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<td>Borderline (uncertain malignant potential)</td>
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<td>Mucinous cystic tumor with moderate dysplasia</td>
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<td>Intraductal papillary mucinous tumor with moderate dysplasia</td>
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<td><strong>Malignant</strong></td>
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<td>Intraductal papillary mucinous carcinoma</td>
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Source.—Reference 5.
Figure 1. Imaging patterns of intraductal papillary mucinous tumor (IPMT) of the pancreas. (a, b) Drawings show main duct IPMT with segmental (a) and diffuse (b) involvement of the MPD. In diffuse involvement, cystic dilatation of branch ducts and dilatation of the major papilla protruding into the duodenal lumen (arrows) are frequently present. (c, d) Drawings show branch duct IPMT with a macrocystic (c) and microcystic (d) pattern.

The age of occurrence is in the 6th decade. The clinical history is variable, but recurrent episodes of acute pancreatitis or dull pain, as seen in patients with chronic pancreatitis, may be elicited from patients with careful questioning. Rarely, IPMT manifests as new-onset diabetes; frequently, IPMT is detected with cross-sectional imaging performed for evaluation of nonspecific abdominal pain.

- **Segmental Involvement of the MPD**
  In the early stages of IPMT with segmental involvement of the MPD, the adjacent pancreatic parenchyma is normal or thin. At cross-sectional imaging, it is difficult to differentiate this appearance from that of localized (segmental) chronic obstructive pancreatitis (Fig 2). In such
Main duct IPMT with segmental involvement of the pancreatic body and tail. Contrast material-enhanced CT scans (presented from cephalic [a] to caudal [b]) show dilatation of the MPD within the pancreatic body with atrophy of the surrounding parenchyma. The dilated duct communicates with an area of cystic ectasia (*). The area of ectasia resembles a cystic tumor, but the communication with the MPD allows correct diagnosis. The pancreatic head is of normal size. The common bile duct (arrowhead) and the cephalic segment of the MPD (arrow) are normal. Partial pancreatectomy was performed.

Occasionally, segmental ductal dilatation evolves into a cystic appearance (Figs 4, 5). When the lesion is localized to the pancreatic body or tail, the remainder of the pancreas is normal (Fig 4). Conversely, if the tumor is located in the pancreatic head, upstream dilatation of the MPD results (Fig 5a, 5b). In cases with a cystic appearance (Figs 4, 5), the tumor resembles a peripheral mucinous cystic tumor (cystadenoma, cystadenocarcinoma).
Figure 5. Main duct IPMT with segmental involvement of the pancreatic head. (a, b) Axial ultrasonographic (US) scans show a dilated MPD (*) within the pancreatic body; there is a direct communication (arrow) between the dilated MPD and an area of cystic ectasia in the cephalic duct (arrowheads). The area of ectasia has sharp margins and inhomogeneous low-level internal echoes. (c) CT scan shows that the cystic lesion is interposed between the duodenum (d) and superior mesenteric vein (arrowhead) and is thin walled. A soft-tissue nodule is seen along the left margin of the lesion (arrow). (d) Axial T2-weighted spin-echo magnetic resonance (MR) image (repetition time msec/echo time msec = 2,000/90) shows overall high signal intensity of the lesion and lower signal intensity of the solid nodule along the left margin (arrow). Pancreatoduodenectomy was performed.

cystadenocarcinoma). However, when a peripheral mucinous cystic tumor is present, the MPD is almost always normal. On the contrary, an IPMT with a cystic appearance causes upstream MPD dilatation (Fig 5a, 5b). These lesions demonstrate variable thickness of the peripheral wall. The wall can be so thick that the lesion may mimic a necrotic ductal adenocarcinoma. Thick walls and solid mural nodules correlate with malignant potential of the lesion (11).

The imaging diagnosis can be made with greater certainty when filling defects are demonstrated inside the ductal segment involved by the tumor (Fig 5c, 5d) (9,12). Filling defects appear hyperechoic at US, hyperattenuating at CT, and hypointense relative to the surrounding fluid at T2-weighted MR imaging. Mural nodules and mucin globs are difficult to differentiate. The relationship of the filling defect to the ductal wall has to be demonstrated, with mural nodules being clearly attached to the wall (Fig 5c, 5d). Conversely, mucin globs are located in the gravity-dependent portion of the duct or isolated from the wall. Moreover, at CT and MR imaging, mural nodules enhance after contrast material administration, whereas mucin does not. Nevertheless, ERCP remains the imaging modality of choice for diagnosis. In fact, ERCP easily demonstrates direct communication of the cystically
Diffuse Involvement of the MPD

When IPMT involves the full length of the MPD, without cystic dilatation, differentiation from chronic pancreatitis may be impossible (16,17). The lesion contents are usually homogeneous (Fig 7) and are hypoechoic at US (18), hypoattenuating at CT (9), hyperintense at T2-weighted MR images. In such cases, ERCP is not indicated as it does not provide additional information. For tumors of any size originating from the MPD, total resection should be performed (15). Local resection (ie, the Whipple operation, modified Whipple operation, or partial pancreatectomy) is sufficient when the IPMT is localized to a segment of the pancreas. When the lesion is in the pancreatic head and the MPD is diffusely dilated upstream, it is impossible to differentiate between diffuse invasion of the MPD and simple ductal dilatation related to mechanical obstruction (Fig 6). Analysis of frozen sections from the surgical margin allows one to establish whether the pancreatic resection needs to be extended.
Figure 7. Main duct IPMT with diffuse involvement. (a, b) CT scans (presented from cranial [a] to caudal [b]) show marked dilatation of the MPD, along with dilatation of the branch ducts in the uncinate process (white dot) and pancreatic tail (*). Severe parenchymal atrophy is present. Cystlike dilatations of the branch ducts are bounded by thin septa. Filling defects are not appreciable. (c) Axial T2-weighted spin-echo MR image shows overall high signal intensity within the MPD and the dilated branch ducts (•, *). (d) Contrast-enhanced axial T1-weighted gradient-echo MR image shows low signal intensity within the MPD and the dilated branch ducts. Again, no filling defects are seen. The diagnosis of borderline malignant mucinous tumor was made with fine-needle aspiration biopsy, but surgery was not performed due to the patient's age (74 years).

weighted MR imaging, and hypointense at T1-weighted MR imaging (13,14,19). The presence of IPMT leads to the same changes in the pancreatic parenchyma as in obstructive chronic pancreatitis (16,20). Progressive MPD dilatation produces parenchymal atrophy, which is first localized, then becomes progressively diffuse (Fig 7), significant, and uniform (10). The dilated MPD appears to be bordered with an exquisitely thin wall (9,10). Nevertheless, there are features at cross-sectional imaging that may narrow the differential diagnosis. Dilatation of branch ducts is frequently encountered in association with diffuse dilatation of the MPD and is preferentially located in the uncinate process and pancreatic tail (Figs 7, 8). The presence of
mural nodules or mucin globs (Fig 8) is of critical importance in making the correct diagnosis, in particular when other signs are absent. If malignant degeneration occurs, the solid component is usually readily evident (Fig 9). A finding virtually pathognomonic of IPMT is dilatation of the major papilla, minor papilla, or both with bulging into the duodenal lumen. This finding is well demonstrated with CT and MR imaging (Figs 9b, 10a), particularly when distention of the duodenal lumen with orally administered contrast material is achieved.

In advanced disease, when IPMT causes mass effect in the pancreatic head, obstruction of the common bile duct results from impacted mucin or the tumor itself. A pancreatobiliary fistula is demonstrable in a later stage (9,20,21). The mass in the pancreatic head also indents the duodenal wall. A pancreatoduodenal fistula may result (21).

In the late stage of the disease, tumor dissemination within the peritoneum or retroperitoneum occurs (Fig 9), and localized foci that are slightly hyperechoic at US and slightly hyperattenuating at CT can be recognized on the peritoneal surface (pseudomyxoma peritonei) (Fig 9b). The same findings are encountered in patients with postoperative recurrence. Direct invasion of adjacent viscera may occur, but lymphadenopathy or distant metastases are rarely encountered.

ERCP easily demonstrates protrusion of a dilated papilla or a pancreatoduodenal fistula (Fig 10b) during the endoscopic phase (9,10,21–23).
Moreover, diagnosis of IPMT with ERCP is certain when jellylike mucin leaking from the papilla is demonstrated, even if opacification of the ductal tree is not achieved (9,24). ERCP is limited when adequate injection of contrast material into the pancreatic duct cannot be achieved. In fact, complete opacification of the pancreatic duct and cystic tumors is often difficult because enlargement of the papillary orifice allows reflux of contrast material despite use of a balloon catheter (14). In addition, viscous mucin or intraductal papillary excrescences may prevent full ductal evaluation (10). In such cases, MR cholangiopancreatography is superior to ERCP (13,14) because MR cholangiopancreatography can consistently demonstrate the extension of the IPMT. With maximum-intensity-projection reconstruction, small filling defects might be obscured if a too thick slab of tissue is incorporated into the reconstruction (19). Thus, when IPMT is evaluated with MR cholangiopancreatography, careful review of the source images is necessary.

Demonstration of a dilated MPD with associated ectasia of the branch ducts in the region of the uncinate process and pancreatic tail (Figs 7, 8) is sufficient reason for total pancreatectomy to be performed after histologic confirmation of malignancy with analysis of frozen sections. When cross-sectional imaging reveals spread of the IPMT into the retroperitoneum (Fig 9), any attempt at resection is not reasonable (15).

### BRANCH DUCT IPMT

IPMT arising within a branch duct was described in 1986 (8). These lesions are more easily recognized than main duct IPMT because they appear as masses (9). Once detected, the lesion must be differentiated from other cystic pancreatic masses, and the aggressive potential of the lesion should be assessed (11).

Branch duct IPMT occurs with equal frequency in male patients and female patients. Patients with branch duct IPMT tend to be older than those with main duct IPMT (8). In our experience with 56 cases of histologically proved IPMT, the mean age was 63.4 years (range, 37–76 years) for patients with the branch duct form versus 57.2 years (range, 34–75 years) for patients with the main duct form. When small, the lesion usually comes to clinical attention as an incidental finding in patients undergoing imaging for other abdominal conditions. Although the symptoms are nonspecific, patients may present with symptoms mimicking those of acute or chronic pancreatitis.

Branch duct IPMT is most frequently located in the uncinate process (9,25,26) but can be found throughout the pancreas, particularly in the pancreatic tail (16,27). The tumor may demonstrate a macrocystic (Fig 1c) or microcystic (Fig 1d) pattern.
In the early stage, regardless of the intrapancreatic location, branch duct IPMT appears as round or oval, small, lobulated masses at imaging (Figs 11–14). In this stage, the MPD is usually normal (Figs 11, 13, 14) or slightly dilated (Fig 12). The communication of the tumor with the MPD can occasionally be demonstrated with thin-section spiral CT (Figs 11b, 11c, 12a–12c). The administration of intravenous secretin enhances detection of the communication be-
mentioned earlier in regard to main duct IPMT, sometimes mucin may prevent adequate inflow of contrast material into the cystically dilated branches (Fig 12d).

Figure 12. Branch duct IPMT of the uncinate process. (a–c) CT scans (presented from cranial [a] to caudal [c]) show ectasia of branch ducts (*) within the uncinate process and dilatation of the MPD (white arrow). Black arrow = common bile duct. (d) ERCP image shows the dilated MPD, which contains a filling defect (arrowhead) attributed to mucin. The thick mucin prevents the contrast material from filling the dilated branch ducts seen on the CT scans. Pancreatoduodenectomy was performed.

between the lesion and the MPD (Fig 11b, 11c). The secretin effect and the resulting ductal distention are demonstrable with all imaging modalities, particularly MR cholangiopancreatography. In some cases, only ERCP allows definitive demonstration of the communication between the MPD and the lesion (Fig 13d). As
Figure 13. Branch duct IPMT of the uncinate process. (a) CT scan shows a unilocular, thin-walled, lobulated lesion (*). The MPD is normal. (b) Axial T2-weighted spin-echo MR image shows a hyperintense lesion (*) with the morphologic features seen on the CT scan. (c) Coronal T2-weighted fast spin-echo MR image shows the lobulated contour and unilocular, macrocystic architecture of the lesion more clearly. The lesion partially overlaps the slightly dilated common bile duct. (d) ERCP image shows the dilated common bile duct and passage of contrast material into the lesion, which is in the uncinate process. Pancreatoduodenectomy was performed.

Figure 14. Multifocal branch duct IPMT. (a) CT scan shows multiple intrapancreatic and peripancreatic cyst-like lesions (*) involving the whole gland. Total pancreatectomy was performed. (b) Radiograph of the resected specimen shows multifocal ductal ectasia from the head to the tail of the gland.
Figure 15. Branch duct IPMT of the uncinate process. (a, b) CT scans (presented from cranial [a] to caudal [b]) show marked dilatation of the MPD and parenchymal atrophy. A multilocular, microcystic lesion is visible in the uncinate process as well. The tumor compresses the duodenal lumen. (c, d) Gadolinium-enhanced coronal T1-weighted gradient-echo MR images (presented from anterior [c] to posterior [d]) show the internal microcystic architecture of the lesion (arrows) more clearly. Pancreatoduodenectomy was performed; the frozen section from the surgical margin was free of tumor.

In later stages, the lesion may seed the MPD, and the typical findings of main duct IPMT are then demonstrated. These findings include diffuse dilatation of the MPD along with diffuse dilatation of branch ducts and bulging of the papilla into the duodenal lumen (Figs 15–17). The MPD involvement produces all of the parenchymal changes seen in exclusively main duct IPMT. In advanced disease, obstruction of the common bile duct and mass effect along the duodenal
loop occur (Fig 17). Tumor growth and MPD involvement usually require a long time to develop, but rarely faster growth occurs (Fig 18).

Branch duct IPMT occurs in two macroscopic patterns (6). The microcystic pattern is characterized by multiple thin septa separating fluid-filled lacunae (Fig 15) and mimics a serous cystadenoma (9). Demonstration of a communication between the lesion and the MPD, which is frequently dilated, leads to the correct diagnosis; this finding can be seen with ERCP and occasionally with MR cholangiopancreatography or CT. The macrocystic pattern, which is much more frequent, is characterized by a unilocular or multilocular internal architecture, with the multilocular architecture related to the presence of sparse septa (Figs 11–14, 16, 17) (26,28). In some cases, filling defects that represent mucin globs or papillary projections of tumor are recognizable. When the nature of the filling defect is in doubt, the differential diagnosis is sometimes facilitated by changing the position of the patient. If the cystic space is large enough, mobility of the mucin is easily demonstrated with US or CT (Fig 16). Differentiation of these forms of IPMT from other cystic tumors, especially mucinous cystadenoma, is possible owing to the demonstration of a communication with the main duct, which is rarely present in mucinous cystadenoma. Differentiation of IPMT from pseudocysts is possible because of the presence of filling defects related to mucin deposition or papillary proliferation in IPMT.

**Figure 16.** Branch duct IPMT. (a–c) CT scans (presented from cranial [a] to caudal [c]) show a multilocular, macrocystic lesion in the uncinate process and dilatation of the MPD. Inside the main cyst, a filling defect is visible in the gravity-dependent portion (arrow). (d) CT scan obtained with the patient in the prone position shows that the defect has moved to the opposite side (arrow), a finding indicative of inspissated mucin. Pancreatoduodenectomy was performed.
Figure 17. IPMT of uncertain origin but presumably originating from branch ducts of the uncinate process. (a) CT scan shows significant dilatation of the MPD and a conspicuous mass of fluid and soft-tissue attenuation that involves the pancreatic head and uncinate process. The lesion seems to involve the mesenteric vessels. A tiny calcification is seen in the MPD at the level of the pancreatic body (arrow). The gastric antrum is pushed ventrally (*). (b) Gadolinium-enhanced axial T1-weighted gradient-echo MR image clearly shows the focal lesion and the MPD dilatation upstream. Centrally located liquid lacunae, gross septa, and intraluminal nodules are seen. Exploratory laparotomy was performed.

Figure 18. Branch duct IPMT of the pancreatic body. (a) CT scan shows a normal-sized pancreas with multifocal slight ectasia of the branch ducts and slight dilatation of the MPD. (b) CT scan obtained 8 months later shows more severe dilatation of the branch ducts and of the involved MPD segment. (c) Intraoperative pancreatogram shows passage of contrast material from the MPD to the ectatic branch ducts (arrows). The dilated branch ducts demonstrate mass effect at the level of the pancreatic body and tail.
The thickness of the tumor wall and septa is variable. In benign lesions, both the wall and the septa are regular and thin (Figs 11–14). In malignant lesions, the wall appears irregular and thick, as do the septa, which demonstrate solid nodules (Figs 15–17).

In cases of branch duct IPMT, total resection should be performed when dilatation of the MPD is present, even if the lesion is very small. Branch duct IPMTs less than 2.5 cm in diameter with a thin wall and a normal MPD may be monitored with serial imaging studies. These lesions are almost always benign (hyperplasia) and grow very slowly or not at all (10,29,30). Therefore, once the intraductal origin of the lesion has been established (noninvasively or with ERCP), US or CT and MR cholangiopancreatography are the imaging modalities of choice for documenting the stability of the lesion.

**CONCLUSIONS**

In advanced-stage IPMT, US may suggest the diagnosis by demonstrating a diffusely dilated MPD (main duct IPMT) or a large mass in the uncinate process (branch duct IPMT). Thin-section contrast-enhanced CT allows confirmation of the diagnosis and demonstrates the extent of the tumor, thus directing clinical decision making. According to the clinical (as opposed to imaging) literature, ERCP represents the standard of reference for diagnosis of IPMT. However, in advanced-stage tumors, ERCP has significant limitations. Reflux of contrast material due to an excess of mucin or a patent papillary orifice hinders filling of the ductal tree (10,31).

As small (<2.5-cm-diameter) IPMTs are more frequently encountered, the question of the characterization and most appropriate management of early-stage tumors arises (11,29,32). Branch duct IPMTs are usually easily characterized, at least with ERCP. The sensitivity of US, CT, and MR imaging allows minimal ectasia of the MPD to be identified. In this case, the real issue is the significance of the abnormality. Inflammatory obstruction (previous acute pancreatitis) (16) or neoplastic obstruction (small ductal adenocarcinoma) (33), as well as IPMT, may be responsible for segmental dilatation of the MPD. When the cause of the obstruction is not identified, only ERCP is reliable for characterizing the lesion by demonstrating (a) features consistent with inflammatory or neoplastic obstruction or (b) a communication between the MPD and the dilated ductal segment where the IPMT is located. In these cases, filling defects resulting from mucin or tumor are important clues to the correct diagnosis. MR cholangiopancreatography adequately demonstrates the segmental dilatation of the MPD (14) but does not always show the communication between the pancreatic duct and the lesion. In the future, secretin-enhanced MR cholangiopancreatography may improve the detectability of this crucial pathologic anatomic feature.

**REFERENCES**