Pancreatic Incidentalomas: Review and Current Management Recommendations

Binit Sureka¹ Vaibhav Varshney²

¹Department of Diagnostic and Interventional Radiology, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India
²Department of Surgical Gastroenterology, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India

Address for correspondences Binit Sureka, MD, DNB, MBA, Department of Diagnostic and Interventional Radiology, All India Institute of Medical Sciences, Basni Industrial Area, MIA 2nd Phase, Basni, Jodhpur 342005, Rajasthan, India (e-mail: binit sûrékapgi@gmail.com).

Abstract
There has been significant increase in the detection of incidental pancreatic lesions due to widespread use of cross-sectional imaging like computed tomography and magnetic resonance imaging supplemented with improvements in imaging resolution. Hence, accurate diagnosis (benign, borderline, or malignant lesion) and adequate follow-up is advised for these incidentally detected pancreatic lesions. In this article, we would review the various pancreatic parenchymal (cystic or solid) and ductal lesions (congenital or pathological), discuss the algorithmic approach in management of incidental pancreatic lesions, and highlight the key imaging features for accurate diagnosis.

Keywords
► duct
► incidentaloma
► pancreas
► pancreatic cyst

Introduction
The term “pancreatic incidentaloma” (PI) refers to those lesions in the pancreas that are diagnosed incidentally when obtaining an imaging study of the abdomen not intended to look for a pancreatic pathological process.¹ This phenomenon is not new to the pancreas and has been quite often observed in other organs such as adrenal glands, thyroid, parathyroid, pituitary, liver, prostate, and kidneys. The first case of PI was described by Kostiuk in 2001.² Incidental pancreatic cysts are frequently encountered now due to advancements and easy availability of imaging. The reported incidence of incidental cystic pancreatic lesions varies, depending on the imaging technique used. In computed tomography (CT), the prevalence varies from 0.5 to 3%, whereas in magnetic resonance imaging (MRI), this prevalence increases to 18 to 19.6%.³ ⁴ In a postmortem study by Kimura et al, cysts less than 1 cm in size were detected in 24% of cases.⁵ Recent published studies have shown that the incidence of incidental PI is rising.⁶

When a PI is encountered, the first aim is to classify whether it is a pancreatic parenchymal or ductal lesion. PIs in the pancreatic parenchyma can be either cystic or solid. Incidental lesions in the main pancreatic duct (MPD) can be congenital variations or a pathological process involving the MPD. The second aim is to further classify the lesion into benign or malignant. Various cystic and solid incidentalomas in the pancreatic parenchyma are listed in ➔ Table 1.⁷

Incidental Cystic Pancreatic Tumors
Most incidental cystic pancreatic lesions are benign.⁸ ⁹ The first step is to differentiate these cystic lesions from pancreatic pseudocysts. Serous cystadenoma, mucinous cystic lesions, and intraductal papillary mucinous neoplasms (IPMNs) account for more than 90% of primary cystic pancreatic tumors.¹⁰ Most of the patients are asymptomatic at the time of presentation. Symptomatic patients may present with abdominal pain, jaundice, weight loss, and/or recurrent episodes of pancreatitis. Morphological classification of cystic lesions of the pancreas is given in ➔ Table 2.¹¹

Unilocular Cysts
Pancreatic pseudocysts are the most commonly encountered unilocular cysts. Others include mucinous and serous cystadenoma, lymphoepithelial cysts, retention cyst, developmental cyst, epidermoid cyst in intrapancreatic spleen, endometrial cyst, and cystic neuroendocrine tumor or an infectious cyst (➔ Figs. 1 and 2). A unilocular lesion in...
Pancreatic Incidentalomas
Surekha, Varshney
Annals of the National Academy of Medical Sciences (India) Vol. 55 No. 1/2019

A patient with a clinical history of pancreatitis is almost always a pseudocyst. A lobulated unilocular cyst located in the head of the pancreas should raise the suspicion of a serous cystadenoma.

---

Table 1 Pancreatic incidentalomas

<table>
<thead>
<tr>
<th>Exocrine</th>
<th>Infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td>Mycobacterium avium complex</td>
</tr>
<tr>
<td>SCN</td>
<td>Mycobacterium tuberculosis</td>
</tr>
<tr>
<td>MCN</td>
<td>Rare and atypical fungal and viral infections</td>
</tr>
<tr>
<td>IPMN</td>
<td></td>
</tr>
<tr>
<td>Mature cystic teratoma</td>
<td></td>
</tr>
<tr>
<td>Borderline</td>
<td></td>
</tr>
<tr>
<td>Mucinous cystic tumor with moderate dysplasia</td>
<td></td>
</tr>
<tr>
<td>Intraductal papillary mucinous tumor with moderate dysplasia</td>
<td></td>
</tr>
<tr>
<td>SPN</td>
<td></td>
</tr>
<tr>
<td>Malignant</td>
<td></td>
</tr>
<tr>
<td>Ductal adenocarcinoma</td>
<td></td>
</tr>
<tr>
<td>Osteoclastlike giant cell tumor</td>
<td></td>
</tr>
<tr>
<td>Serous cystadenocarcinoma</td>
<td></td>
</tr>
<tr>
<td>Mucinous cystadenocarcinoma</td>
<td></td>
</tr>
<tr>
<td>IPMN</td>
<td></td>
</tr>
<tr>
<td>Acinar cell carcinoma</td>
<td></td>
</tr>
<tr>
<td>Pancreatoblastoma</td>
<td></td>
</tr>
<tr>
<td>Solid pseudopapillary carcinoma</td>
<td></td>
</tr>
<tr>
<td>Ampullary adenocarcinoma</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Endocrine</th>
<th>Nonislet cell tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrinoma</td>
<td>Adenosquamous carcinoma</td>
</tr>
<tr>
<td>Glucagonoma</td>
<td>Anaplastic tumors</td>
</tr>
<tr>
<td>GRF-secreting tumor</td>
<td>Colloid carcinoma</td>
</tr>
<tr>
<td>Insulinoma</td>
<td>Granulocytic sarcoma</td>
</tr>
<tr>
<td>PP-secreting tumor</td>
<td>Leukemia</td>
</tr>
<tr>
<td>Somatostatinoma</td>
<td>Lymphoma</td>
</tr>
<tr>
<td>VIPoma</td>
<td>Primitive neuroendocrine tumor</td>
</tr>
<tr>
<td>Serotoninoma</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cystic lesions</th>
<th>Inflammatory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign pancreatic cysts</td>
<td>Eosinophilic pancreatitis</td>
</tr>
<tr>
<td>Dysontogenic cysts</td>
<td>Focal pancreatitis</td>
</tr>
<tr>
<td>Hydatid cyst</td>
<td>Inflammatory myofibroblastic tumor</td>
</tr>
<tr>
<td>Cysticercosis</td>
<td>Lymphoid hyperplasia</td>
</tr>
<tr>
<td>LECs</td>
<td>Wegener’s disease</td>
</tr>
<tr>
<td>Pancreatic dermoid cysts</td>
<td>Xanthogranulomatous pancreatitis</td>
</tr>
<tr>
<td>Retention pancreatic cysts</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Congenital</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital cyst</td>
<td></td>
</tr>
<tr>
<td>Epidermoid cyst in IPAS</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Classification according to imaging morphology

<table>
<thead>
<tr>
<th>Unilocular</th>
<th>Microcystic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudocyst</td>
<td>Serous cystadenoma</td>
</tr>
<tr>
<td>Cystic neuroendocrine tumor</td>
<td>Microcystic variant of ductal adenocarcinoma</td>
</tr>
<tr>
<td>Unilocular serous cystadenoma</td>
<td>(very rare)</td>
</tr>
<tr>
<td>Unilocular mucinous cystadenoma</td>
<td></td>
</tr>
<tr>
<td>Retention cyst</td>
<td></td>
</tr>
<tr>
<td>Developmental cyst</td>
<td></td>
</tr>
<tr>
<td>Epithelial cyst</td>
<td></td>
</tr>
<tr>
<td>Lymphoepithelial cyst</td>
<td></td>
</tr>
<tr>
<td>Epidermoid cyst in intrapancreatic accessory spleen</td>
<td></td>
</tr>
<tr>
<td>Endometrial cyst</td>
<td></td>
</tr>
<tr>
<td>Infectious cyst</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Macrocytic</th>
<th>Cystic transformation of the pancreas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucinous cystadenoma</td>
<td>Dysontogenic cyst</td>
</tr>
<tr>
<td>BD-IPMN</td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Oligocystic serous cystadenoma</td>
<td>Disseminated serous cystadenoma</td>
</tr>
<tr>
<td>Lymphangiomatosis</td>
<td>Congenital syndromes such as von Hippel–Lindau’s disease, polycystic kidney disease, Ivemark’s syndrome, trisomy 13 or 15, Meckel–Gruber’s syndrome</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cyst with ductal communication</th>
<th>Multifocal</th>
</tr>
</thead>
<tbody>
<tr>
<td>IPMN</td>
<td>BD-IPMN</td>
</tr>
<tr>
<td>Collections postpancreatitis as a part of disconnected duct syndrome</td>
<td>Pseudocysts</td>
</tr>
<tr>
<td>Retention cyst/squamous cyst</td>
<td>Serous cystadenoma</td>
</tr>
<tr>
<td></td>
<td>Neuroendocrine tumor</td>
</tr>
<tr>
<td></td>
<td>Developmental cyst</td>
</tr>
<tr>
<td></td>
<td>Epithelial cyst</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Solid cystic</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>SPN</td>
<td></td>
</tr>
<tr>
<td>Pancreatoblastoma</td>
<td></td>
</tr>
<tr>
<td>Cystic metastasis</td>
<td></td>
</tr>
<tr>
<td>Cystic degeneration in solid tumors</td>
<td></td>
</tr>
<tr>
<td>Malignant transformation in cystic tumors</td>
<td></td>
</tr>
<tr>
<td>Metastases</td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic pseudocyst</td>
<td></td>
</tr>
</tbody>
</table>

---

Abbreviations: GRF, growth hormone releasing factor; IPAS, intrapancreatic accessory spleen; IPMN, intraductal papillary mucinous adenoma; LEC, lymphoepithelial cyst; MCN, mucinous cystadenoma; PP, pancreatic polypeptide; SCN, serous cystadenoma; SPN, solid pseudopapillary tumor; VIPoma, pancreatic neuroendocrine tumor that secretes vasoactive intestinal peptide.

---

Microcystic Lesions

The most important differential diagnosis of a microcystic lesion in the pancreas is serous cystadenoma. It usually demonstrates a polycystic or microcystic pattern consisting of cysts up to 2 cm in size. They are usually lobulated. The septa and wall may show enhancement (Fig. 3). A stellate pattern of calcification is visible in 30% of the patients and is considered a characteristic of a serous cystadenoma. A rare
Fig. 1 A 43-year-old-female with incidentally detected lymphoepithelial cyst postcholecystectomy in the pancreas. (A) Axial T1-weighted magnetic resonance imaging showing unilocular cystic lesion, which is mildly hyperintense in the head of the pancreas (B) and hyperintense on T2-weighted images (C), showing bright signal on diffusion-weighted imaging (D) with no enhancement on T1-weighted postcontrast gradient echo images.

Fig. 2 A 60-year-old male with incidental cystic pancreatic neuroendocrine tumor. Axial postcontrast T1-weighted image showing incidental unilocular cystic lesion (arrow) in the tail of the pancreas, showing peripheral arterial enhancement suggestive of cystic neuroendocrine tumor.

Fig. 3 A 68-year-old-female with incidental serous cystadenoma. Axial contrast-enhanced computed tomography image showing polycystic lesion in the head of the pancreas (arrow) with thin internal septations in a case of serous cystadenoma.

differential diagnosis of a microcystic lesion in the pancreas is a microcystic variant of ductal adenocarcinoma.

Macroscopic Lesions
Mucinous cystic neoplasms (cystadenomas) and branch-duct (BD) IPMNs usually present as macrocystic lesions. Mucinous cystadenomas mainly involve the body and tail of the pancreas and do not communicate with the MPD. It is important to differentiate serous from mucinous cystic neoplasm as surgery is the treatment of choice in mucinous cystic neoplasm. A peripheral eggshell calcification is highly suggestive of a potentially malignant mucinous cystic neoplasm. Other differential diagnoses of macrocystic lesions in the pancreas are lymphangiomas, lymphoepithelial cysts, infectious cysts, mesothelial cysts, and duplication cysts.

Cyst with Solid Component (Solid Cystic)
Tumors with this morphology are solid pseudopapillary neoplasms, pancreatoblastomas, cystic metastases, cystic degeneration in solid tumors, malignant transformation in cystic tumors, and hemorrhagic pseudocysts. Cysts with...
a solid component can be unilocular or multilocular and
may or may not have ductal communication. Therefore,
true cystic tumors with solid component as well as solid
pancreatic neoplasms with a cystic component or cystic
degeneration are included in this category. Most tumors in
this category are malignant and should be surgically treat-
ed. MR cholangiopancreatography (MRCP) is superior to
single-section helical CT to characterize these tumors.

Cystic Transformation of the Pancreas
Cystic transformation of the pancreas is seen in dysonto-
genetic cyst, cystic fibrosis, disseminated variant of serous
cystadenoma, congenital syndromes such as von Hippel–
Lindau’s disease (VHL), autosomal dominant polycystic kid-
ney disease (ADPKD), Ivemark’s syndrome, trisomy 13 or 15,
Meckel–Gruber’s syndrome, and so on. Polycystic disease
of the pancreas is also known as dysontogenetic cysts or
congenital cysts of the pancreas. It is a very rare entity that
may occur as a solitary cyst, polycystic disease in association
with renal cysts, and liver, central nervous system, or retinal
abnormalities. Pancreatic involvement in VHL is in the form
of simple cysts (71%), serous cystadenomas (15%), pancre-
atic neuroendocrine tumors (pNETs; 10%) and rarely cystic
replacement of the entire pancreas. Kim et al have shown
that pancreatic cysts are five times more prevalent in
patients with ADPKD with PKD2 mutation than in patients
with PKD1 mutation. PKD1 has a more aggressive disease
course, with an earlier age of symptom onset, end-stage
renal disease, and death. Thus, the potential to discriminate
PKD1 from PKD2 on MRI has important prognostic implica-
tions. MRI identification of pancreatic cysts in ADPKD sig-
nificantly increases the likelihood that a PKD2 mutation is
present.

Cyst with Ductal Communication
Tumors included in this subgroup are IPMNs, collections
postpancreatitis as a part of disconnected duct syndrome,
and retention cysts. IPMNs are more common in elderly
males in the sixth to seventh decade of life. Three types of
IPMNs may be observed: main duct, BD, and mixed variant.
Retention cysts are cystic dilatation of the pancreatic duct. It
may or may not be associated with an obstructive cause such
as calculi, stricture, mucin plugs, and tumors.

Multifocal Cystic Lesions
BD-IPMN, pseudocysts, serous cystadenomas, neuroendo-
crine tumors, developmental cysts, and epithelial cysts can
be multifocal and should be considered in the differential
diagnoses of multifocal cystic lesions of the pancreas.

Incidental Solid Pancreatic Tumors
The incidence of benign disease in solid pancreatic tumors
suspicious of cancer ranges from 6 to 21%. Chronic pancre-
atitis presenting as inflammatory pancreatic mass accounts
for almost 70% of the benign lesions, with alcoholic pancre-
atitis being the most common cause (60%) with autoimmune
pancreatitis (AIP) in up to 11% of the patients.

Pancreatic Adenocarcinoma
The most frequent solid lesion in the pancreas is pancreatic
ductal adenocarcinoma (PDAC). Symptomatic patients pres-
ent with advanced disease at the time of diagnosis (extensive
local disease in ~40% and metastases in 40–55%), leaving less
than 20% of patients as candidates for potentially curative
resection. The earliest imaging finding of a PDAC before
a mass becomes apparent is pancreatic ductal dilatation or
pancreatic duct cutoff. On imaging, pancreatic carcinomas
(PCs) are hypovascular, hypoenhancing lesions when com-
pared with the surrounding pancreatic parenchyma. On MRI,
most PDACs are hypointense on unenhanced T1-weighted se-
quences when compared with the surrounding pancreas
and are hypointense or isointense on T2-weighted images.
The sensitivity and specificity of fluoro-2-deoxy-d-glucose–
positron emission tomography (FDG-PET) for the diagnosis
of PC in patients with normal blood glucose levels range from
85 to 100% and 67 to 99%, respectively. Combination of PET
and CT may offer a better accuracy. Serum tumor markers
can be helpful in differentiating benign from malignant
pancreatic masses. The addition of other tumor markers such
as Ca-125 does not increase the diagnostic accuracy of CA19-9
and is the gold standard marker for PDAC with sensitivity and
specificity as high as 87 and 98%, respectively.

Pancreatic Neuroendocrine Tumors
Over the last decade, the wide use of imaging technolo-
gy has led to the rising incidence of pNETs. pNETs are rare
and account for 2 to 4% of all pancreatic neoplasms, with an
incidence of 1.5 in 100,000. In recent years, the detection
of incidentally nonfunctioning pNETs (NF-pNETs) has
rapidly increased due to the widespread use of endoscopic
cross-sectional imaging. Nearly 60% secrete one or more
biologically active peptides, resulting in clinical syndromes.
The most frequent functioning tumors are insulinomas, gas-
trinomas, glucagonomas, VIPomas (a pancreatic neuroen-
docrine tumor that secretes vasoactive intestinal peptide),
and somatostatinomas. Between 30 and 40% of pNETs are

Fig. 4 A 27-year-old female with incidental pancreatic neuroendo-
crine tumor. Axial contrast-enhanced computed tomography image
showing incidental well-defined hypervascular lesion (arrow) in head
uncinate of the pancreas.
nonfunctioning, and this is more likely to be discovered incidentally when symptoms due to the presence of the mass are not yet obvious.\textsuperscript{38}

On CT scan, most pNETs are isodense or moderately hypodense masses showing good arterial enhancement (\texttextbullet{} Fig. 4). Calcification, necrosis, and cystic degeneration seem to be more common in large nonfunctioning tumors or with malignant transformation. MRI has a diagnostic sensitivity of 78 to 91%, which is equivalent to that of dynamic CT.\textsuperscript{39,40} On MRI, pNETs show low signal intensity on T1-weighted images and high signal intensity on T2-weighted images.\textsuperscript{51,42}

The most important imaging differential diagnosis is intrapancreatic spleen, which shows enhancement characteristics similar to those of splenic parenchyma (\texttextbullet{} Fig. 5).

\textbf{Inflammatory Pancreatic Mass}

The combination of acute pancreatitis and cancer is unusual. Pancreatic cancer represents 1 to 2% of acute pancreatitis etiologies, and only 3% of cancers manifest as acute pancreatitis.\textsuperscript{43} The risk of developing pancreatic cancer in patients with chronic pancreatitis is around 15 times higher than in the average population.\textsuperscript{44} Dilatation of the pancreatic duct, and double-duct sign with both biliary and pancreatic obstruction or interruption of the pancreatic duct are unusual and should lead to considering an underlying carcinoma. Sheathing of the celiac trunk and/or mesenteric artery is seen in 30 to 60% of CT scans of adenocarcinoma.\textsuperscript{46} But this can also be seen in AIP or IgG4 (immunoglobulin G4 related) conditions with extrapancreatic manifestations.

Adenocarcinoma developing in the background of chronic pancreatitis is difficult to detect on imaging. In the context of chronic pancreatitis, calcifications displaced by the mass is a pointer suggesting a coexisting PC.\textsuperscript{47,48} Nearly 10% of presumed PCs that are surgically operated have been composed of pseudotumoral forms of pancreatitis, nearly half of which are thought to be focal forms of AIP, involving mainly the head.\textsuperscript{49,50} Certain imaging criteria are helpful in diagnosing AIP, such as both early and delayed homogeneous enhancement of the lesion close to that of normal parenchyma, peripheral pseudcapsule, a duct visible in the mass with an hourglass stenosis, absence of upstream atrophy or marked dilatation of the pancreatic duct (<4 mm), multifocal involvement, absence of vascular involvement, and presence of extrapancreatic manifestations.\textsuperscript{51}

\textbf{Incidental Congenital Main Pancreatic Duct Anomalies}

Congenital anomalies and normal variants of the pancreas and pancreatic duct may not be detected until adulthood and are often discovered as an incidental finding in asymptomatic patients. These anomalies are considered and detected only when patients present with idiopathic pancreatitis. MRCP is the modality of choice nowadays for the assessment of congenital pancreatic anomalies since it depicts ductal anatomy rapidly and noninvasively. Anatomic variations and developmental anomalies of the pancreas and pancreatic duct include variations of the course of the pancreatic duct (descending, sigmoid, vertical, and loop-shaped course), variation of the configuration of the pancreatic duct (bifid configuration with dominant duct of Wirsung [60%], dominant duct of Santorini without divisum [1%], absent duct of Santorini, and ansa pancreatica), duplication anomalies, anomalous pancreatico-biliary ductal junction, pancreas divisum (4–14%), annular pancreas, ectopic pancreas, and pancreatic agenesis and hypoplasia of the dorsal pancreas and accessory pancreatic lobe.\textsuperscript{52,53}

\textbf{Incidental Pathological Processes Involving the MPD}

Genetic mutation associated pancreatitis (GMAP) also sometimes referred to as idiopathic painless chronic pancreatitis can present as an incidental ductal disease process while imaging for symptoms not related to the pancreas. Several gene mutations associated with chronic pancreatitis have been identified, with the most frequent involving the \textit{CFTR} (cystic fibrosis transmembrane regulator) gene, the \textit{SPINK1} (serine protease inhibitor, Kazal type 1) gene, and the \textit{PRSSI1} (cystic fibrosis transmembrane regulator) gene. According to some authors, the patients with pancreatitis associated with one of these gene mutations show onset at a younger age than those with pancreatitis related to other factors, even though the diagnosis is often late compared with the appearance of symptoms. Accurate diagnosis of GMAP is important for a careful follow-up of these patients, as the risk of developing pancreatic adenocarcinoma is higher in this group than in the normal population or in patients affected by chronic pancreatitis not associated with gene mutations. On imaging, typical bull’s eye pattern of stones, with a dense peripheral rim and a noncalcified radiolucent central core with stones greater than 15 mm in size, is seen.\textsuperscript{54,55}

pNETs expressing serotonin (carcinoid tumors) account for a small portion of neuroendocrine tumors.\textsuperscript{56} Segmental changes in the pancreatic duct are being increasingly encountered as patients undergo abdominal imaging for
evaluation of a variety of symptoms. The two most common causes of segmental pancreatic duct dilatation and pancreatic atrophy are chronic pancreatitis and malignant neoplasms such as PDAC. In rare instances, small serotonin secretin neuroendocrine tumors (serotoninoma) can induce fibrogenesis due to production of 5-hydroxyindoleacetic acid and serotonin, leading to obstruction of the pancreatic duct. These tumors are often detected incidentally while imaging patients for symptoms other than pancreatic etiology.

### Management of Incidental Pancreatic Lesions

Most guidelines reach the consensus that the presence of a potentially resectable solid pancreatic mass in a CT scan or endoscopic ultrasound (US) in an otherwise healthy patient, with no clinical or biochemical characteristics suggesting a benign condition, should prompt surgical treatment. Management of incidentally detected pNETs is a debatable topic. Indications for surgery in pNETs are functioning pNET and

**Figure 6** Algorithm for the management of pancreatic incidentalomas.

**Table 3** Practical tips in pancreatic incidentalomas

<table>
<thead>
<tr>
<th>Cysts requiring surgery</th>
<th>Solid Tumors – Resection (80% malignant)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mucinous cystic neoplasm (MCN)</td>
<td>pNET &gt; 3 cm – Surgery</td>
</tr>
<tr>
<td>2. Solid pseudopapillary neoplasm (SPN)</td>
<td>pNET &lt; 2 cm – Enucleation if superficial</td>
</tr>
<tr>
<td>3. MD-IPMN</td>
<td>pNET &lt; 1 cm – Monitoring</td>
</tr>
<tr>
<td>4. Mixed-IPMN</td>
<td></td>
</tr>
</tbody>
</table>

**Resection criteria for cysts**

- Cysts ≥ 4 cm, symptomatic cysts, enhancing mural nodule ≥ 5 mm, main duct diameter > 6 mm, cyst increasing in size > 2 mm/y, elevated CA 19-9

**Resection criteria for BD-IPMN**

- Obstructive jaundice, enhancing solid nodule, MPD > 10 mm, cyst > 4 cm

**Surveillance is advised in (Follow-up every 6-12 months by EUS/MR according to expertise)**

1. BD-IPMN
2. Indeterminate cysts < 3 cm

### Abbreviations

- CEA, carcinoembyronic antigen; CT, computed tomography; EUS, endoscopic ultrasound; IPMN, intraductal papillary mucinous neoplasm; MCN, mucinous cystadenoma; MD-IPMN, main-duct intraductal papillary mucinous neoplasm; MRI, magnetic resonance imaging; pNETs, pancreatic neuroendocrine tumors; SPN, solid pseudopapillary tumor.
NF-pNETs > 2 cm. pNET < 1 cm can be kept on monitoring, and pNETs of 1 to 2 cm should be considered for enucleation if superficial in location.50,61

Various published guidelines for the management of cystic lesions recommend resection of potentially malignant tumors such as mucinous cystadenomas, solid pseudopapillary tumors, and main and mixed type of intraductal papillary mucinous neoplasms and observe benign lesions such as serous cystadenoma (< 4 cm) and BD IPMN.62-66 Close follow-up and surgical consideration are recommended in cases with worrisome features (symptomatic, cytology suspicious for malignancy, enhancing mural nodule < 5 mm, MPD > 6 mm, cyst size increasing > 2 mm/year, elevated CA19-9).62-66 The International Association of Pancreatologists recommends surveillance for simple cysts < 3 cm in size. Patients with cysts < 1 cm are imaged at an interval of 2 to 3 years (CT/MRI), those with cysts 1 to 2 cm in diameter at an interval of 1 year (CT/MRI), and those with cysts of size 2 to 3 cm at an interval of 3 to 6 months (preferably endoscopic US).64 An algorithmic approach to the management of PIs is illustrated in ►Fig. 6.

Conclusion

PIs are increasingly encountered by the radiologists today and are worrisome. These can be cystic or solid. An incidentally discovered pancreatic cystic lesion without any evidence of worrisome features and of < 2 cm is highly unlikely to cause morbidity or mortality. Around half of these lesions may eventually grow to be larger than 2 cm; therefore, adequate follow-up is advised. Solid PIs > 1 cm are potentially considered malignant, unless proven otherwise, and resection or enucleation is recommended. Key practical tips for the radiologists when dealing with PIs are given in ►Table 3.

Conflict of Interest

None declared.

References

Pancreatic Incidentalomas  Surekha, Varshney 13


