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Review

Pancreatic tumors imaging: An update

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ABSTRACT

Currently, ultrasound (US), computed tomography (CT) and Magnetic Resonance imaging (MRI) represent the mainstay in the evaluation of pancreatic solid and cystic tumors affecting pancreas in 80–85% and 10–15% of the cases respectively. Integration of US, CT or MR imaging is essential for an accurate assessment of pancreatic parenchyma, ducts and adjacent soft tissues in order to detect and to stage the tumor, to differentiate solid from cystic lesions and to establish an appropriate treatment. The purpose of this review is to provide an overview of pancreatic tumors and the role of imaging in their diagnosis and management.

In order to a prompt and accurate diagnosis and appropriate management of pancreatic lesions, it is crucial for radiologists to know the key findings of the most frequent tumors of the pancreas and the current role of imaging modalities.

A multimodality approach is often helpful. If multidetector-row CT (MDCT) is the preferred initial imaging modality in patients with clinical suspicion for pancreatic cancer, multiparametric MRI provides essential information for the detection and characterization of a wide variety of pancreatic lesions and can be used as a problem-solving tool at diagnosis and during follow-up.

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1. Introduction

Currently, ultrasound (US), computed tomography (CT) and Magnetic Resonance imaging (MRI) represent the mainstay in the evaluation of pancreatic solid and cystic tumors affecting pancreas in 80–85% and 10–15% of the cases respectively [1,2]. Integration of US, CT or MR imaging is essential for an accurate assessment of pancreatic parenchyma, ducts and adjacent soft tissues in order to detect and to stage the tumor, to differentiate solid from cystic lesions and to establish an appropriate treatment. The purpose of this review is to provide an overview of pancreatic tumors and the role of imaging in their diagnosis and management.

2. Classification

Pancreatic tumors including a heterogeneous group of primary lesions: adenocarcinoma, neuroendocrine tumor (NET), pancreatic cystic neoplasms, solid pseudopapillary tumor, pancreatoblastoma, pancreatic lymphoma and rare miscellaneous neoplasms [1] (Table 1).

Pancreatic ductal adenocarcinoma (PDA) represents 85–95% of all pancreatic solid pancreatic malignant neoplasms while neuroendocrine tumors are frequently benign and include insulinoma, gastrinoma, glucagonoma, somatostatinoma, vasoactive intestinal polypeptide tumor (VIPoma), Pancreatic polypeptide secreting tumors (PPomas) and non-functioning tumors, amounting to 3–4% of the cases [1].

3. Clinical presentation

Early pancreatic cancer is often asymptomatic. Tumors in the

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Table 1
Pancreatic tumors and tumor-like lesions.

Tumor lesions				Tumor-like lesions
Primitive			Secondary (from)	Focal pancreatitis
Solid exocrine tumors	Solid neuroendocrine tumors (NETs)	Cystic lesions		Fatty infiltration-replacement
Ductal adenocarcinoma	Insulinoma	Intraductal papillary mucinous neoplasm (IPMN)	Renal cell carcinoma	Pseudocysts
Acinar cell carcinoma	Gastrinoma	Serous cystoadenoma	Lung carcinoma	Intrapancreatic accessory spleen
Pancreatoblastoma	Glucaconoma	Mucinous cystic neoplasm	Breast carcinoma	Hydatid cysts
Solid pseudopapillary neoplasm	Vipoma	True cyst	Colorectal carcinoma	Fibrocystic disease
Pancreatic lymphoma	Pancreatic polypeptide secreting tumors (PPoma)	Cystic variants of solid tumors (e.g. Cystic teratoma, Cystic ductal adenocarcinoma, Cystic NET)	Melanoma	Duplication cysts and retention cysts
Miscellaneous carcinomas	Somatostatinoma		Ovarian cancer	Sarcoidosis
	Non-functioning tumors		Sarcoma	Castleman disease

pancreatic head (75% of the cases), often present early with biliary obstruction. However, tumors in the body and tail can remain asymptomatic till late in disease stage [3].

Weight loss, poor appetite, abdominal discomfort, abdominal or midback pain and obstructive jaundice and related symptoms are relatively common and generally occur late in the clinical development; pancreatitis is less common as presenting symptoms [1,4,5]. Digestive problems, nausea and vomiting occur more frequently when the cancer presses on the stomach. Rarely, pancreatic cancers cause diabetes due to the destruction of insulin-making cells. Encasement of vascular structures, infiltration of adjacent bowel and superior mesenteric vein thrombosis may all occur later.

PDA is associated with several rare paraneoplastic syndromes: Trousseau syndrome is traditionally defined as migratory thrombophlebitis [6,7]. Panniculitis is associated with acinar cell carcinoma in 8% of cases; eczematous dermatitis, fibrous cutaneous hand changes, plantar keratoderma, polymyositis, neurological and hematologic manifestation represent other paraneoplastic syndromes [1,8–10].

Signs and symptoms of pancreatic functioning NET are different and dependent on an excessive secretion of hormones. Insulinoma (50%) reveals itself with hypoglycemic attacks featuring neuroglycopenia and sympathetic over-stimulation, including weakness, confusion, sweating, and rapid heartbeat, and/or atypical seizures [1,11–13]. Gastrinomas (20%) produce too much gastrin, causing a condition known as Zollinger-Ellison syndrome, resulting in peptic ulcers which can cause pain, nausea, loss of appetite and anemia [1,11,14–17]. VIPomas (3%) make vasoactive intestinal peptide (VIP) and result in watery diarrhea and hypokalemia [1,11,12,18]. Glucagonomas (1%) produce glucagon that increases glucose levels in the blood; most of the symptoms are often nonspecific, as diarrhea, weight loss, malnutrition and rarely hyperglycemia. The most distinctive feature of a glucagonoma is necrolytic migratory erythema, a red rash with swelling and blisters that often travels place to place on the skin [1,11,12,19]. Somatostatinomas (<1%) produce somatostatin; symptoms can include diarrhea, steatorrhea, nausea, poor appetite and weight loss, gallstones, and symptoms of diabetes [1,11,20]. Ppomas cause an increase in the production of pancreatic polypeptide (PP), but they are rare and have not been associated with any clinical syndrome [21]; some patients also get watery diarrhea.

Signs and symptoms of non-functioning neuroendocrine tumors are caused by mass effect (mainly jaundice, belly pain and weight loss) [11].

Moreover, asymptomatic cancer can be incidentally detected on abdominal scans obtained for other reasons.

4. Imaging

4.1. Plain radiograph

Plain abdominal radiograph has a very limited role in imaging of the pancreas; sometimes it can show coarse parenchymal calcification of the pancreas in 25–59% of patients with chronic pancreatitis; however, calcifications near the pancreas can be confused with splenic artery calcifications.

4.2. Ultrasound

Ultrasound (US) is usually limited in the evaluation of pancreas due to body habitus (adipose tissue) and the interposed intestinal and gastric bloating [22–24]. However, US is the first non-invasive imaging test for the evaluation of pancreas. Abdominal conventional US allows to assess size, site and echogenicity of pancreatic lesions and to evaluate the Wirsung duct caliber ;with a sensitivity and specificity respectively of 75% [24] and an accuracy of 50–70% [25]. Most focal pancreatic lesions are hypoechoic compared to normal parenchyma. Typically dilatation of the common bile duct and pancreatic duct (double duct sign), which is very suggestive for a mass in the pancreatic head, even in the absence of a visible mass, is seen in patients with a pancreatic head tumor.

Endoscopic US (EUS) provides ultra-high resolution images and is commonly accepted as the most sensitive technique for detection of small pancreatic head tumors (<2 cm) [26,27].

4.3. Contrast-enhanced US

The introduction of microbubble contrast agents has improved the diagnostic accuracy of US in the study of pancreatic pathologies [28,29]. Contrast-enhanced US (CEUS) is a cost-effective real-time method that allows the evaluation of the enhancement of pancreatic lesions during the dynamic phases [29] and provides useful findings for differentiating pancreatic carcinoma from chronic focal pancreatitis [30]; moreover CEUS is very accurate in demonstrating NET vascularisation [31].

Even if the Authors themselves suggest that CEUS is an accurate method for the characterization of pancreatic masses [32], CEUS is not sufficient to characterize the tumor, but rather it can improve the accuracy of US of pancreatic lesions incidentally detected as

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