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Pearls and pitfalls of imaging metastatic disease from pancreatic adenocarcinoma: a systematic review

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ABSTRACT

Pancreatic adenocarcinoma is a systemic disease due to the presence of metastatic disease at the time of diagnosis and local recurrence as well as distant metastatic disease after treatment in a majority of patients. Recognition of these metastatic sites may help in accurate staging and assessment of therapeutic response. The authors discuss and illustrate imaging findings of metastatic disease from pancreatic adenocarcinoma in different organ systems with emphasis on entities that can mimic metastatic pancreatic cancer.

1. Introduction

Pancreatic ductal adenocarcinoma is one of the most aggressive malignancies, ranking fourth among causes of cancer-related deaths in the Western world [1]. Metastatic pathways include direct extension of the tumor to the adjacent organs, hematogenous spread, and involvement of the regional lymph nodes. Accurate staging of pancreatic cancer allows assessment of prognosis, stage-specific treatment, and clinical outcomes. Although the 5-year survival rate after primary surgical resection along with adjuvant therapy is over 20% [2,3], this option can only be considered in up to 25% of all patients due to the presence of locally advanced or metastatic disease at the time of diagnosis [4,5]. In surgically unresectable patients, palliative chemotherapy has been the standard treatment, and some of the newer therapy options have shown to improve survival [6,7]. With improved survival benefits from adjuvant chemotherapy [4,5] and the increasing reliance on imaging for assessment of treatment response, there has been an increase in focus on identification of metastatic disease beyond the common sites of disease spread, i.e., the perivascular and hepatic locations. Furthermore, about 70% of all patients develop recurrence and metastatic disease after successful surgical resection [8]. Therefore, the role of imaging is critical not only in staging disease for surgical resection but also to identify the presence of metastatic disease in guiding treatment and assess response to therapy in surgical and nonsurgical patients. Computed tomography (CT) is widely used for staging pancreatic cancer and for the assessment of treatment response and disease surveillance. In this article, the authors present a systematic review of CT findings of sites and patterns of metastases from pancreatic adenocarcinoma in an effort to help radiologists widen their search patterns for accurate disease staging and assessment of treatment response. Although distant organ metastases without local extension in the perivascular and hepatic locations are rare, evaluation of areas beyond the peripancreatic region is important for a number of reasons. First, in the presence of equivocal findings in the perivascular and hepatic locations, a more unequivocal metastatic site elsewhere can provide a definitive evaluation of the disease state. Second, treatment methods may be altered in the presence of new disease. In addition, knowledge of the imaging pitfalls of a number of benign tumors, inflammatory diseases, and other malignancies that mimic metastatic pancreatic cancer is important to help radiologists make the correct diagnosis and avoid overstaging the malignancy.

1.1. CT imaging technique

Routine CT protocol for pancreatic imaging at our institution involves a dual-phase (i.e., arterial and venous phases) cross-sectional imaging with thin slices using multidetector CT. Axial images of the abdomen are acquired at 25–30 s (arterial phase) and 60 s (portal venous phase) after administration of intravenous (IV) contrast using 100–120 ml of iodinated contrast (Visipaque 320 or Omnipaque 350; GE Healthcare, Waukesha, WI, USA). Arterial phase is replaced by the pancreas parenchymal phase at 40 s at many centers. Water as negative oral contrast is used. Positive oral contrast may be administered when there is a question of an enteric leak but is generally not used as it causes significant streak artifact which may hamper the 3-dimensional (3D) postprocessing. The injection rates are routinely 4–5 ml/s with scan

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delays of 25–30 s (arterial phase) and 60 s (portal venous phase). CTs include both thin (0.75 to 1.5 mm) and thick (3 to 5 mm) image series, sagittal and coronal reformatted image series, and 3D postprocessed images. The maximum intensity projection images are valuable in detection of subtle vascular complications such as pseudoaneurysms or occult bleeding sites. Volume-rendered images may help assess anastomotic sites.

2. Metastatic sites

2.1. Perivascular disease

Pancreatic cancer originates in the retroperitoneum and frequently infiltrates the mesentery along the celiac axis and superior mesenteric vessels. Due to the absence of a capsule, local spread occurs early. Vascular invasion is relatively common and is found in 21%–64% of patients with pancreatic adenocarcinoma. It is a key aspect in the evaluation for resectability in the absence of metastatic disease [9]. According to the National Comprehensive Cancer network, tumor encasement of the arteries such as the celiac axis, superior mesenteric artery (SMA), or hepatic artery constitutes contraindication to surgery, while surgery may be performed in the presence of isolated involvement of smaller branches such as the gastroduodenal artery. Surgery may be performed in borderline or marginal resectable disease where there is short-segment venous involvement with suitable vessel above and below the point of involvement for resection and reconstruction, tumor abutment of the celiac axis and SMA, and short-segment encasement of the hepatic artery with sparing of the origin from the celiac axis [10,11]. In such cases, presurgical chemoradiation has also been shown to enhance resectability and inhibit local recurrence, thus improving overall survival [12]. The SMA is most frequently involved in the presence of tumor arising from the head, uncinate process, or the body of the pancreas due to their close proximity [13]. Perivascular sheath-like soft tissue thickening in the presence of an unresected pancreatic cancer is highly suspicious for metastatic disease. A number of nonneoplastic pathologies affecting the peritoneum can also present as soft tissue masses that mimic perivascular spread of disease. Despite long duration and severe obstruction, acute pancreatitis is an uncommon first presentation of pancreatic carcinoma. Pancreatic carcinoma may, however, present initially as acute pancreatitis in up to 17% of patients without gallstone, alcohol abuse, or Endoscopic retrograde cholangiopancreatography (ERCP) as an etiology [14]. Mild peripancreatic stranding due to superimposed inflammation may obscure detection of perivascular disease and may extend to the perivascular regions mimicking tumor spread. Therefore, correlation with patient's symptoms and serum amylase and lipase values is important, and repeat imaging may be necessary after resolution of the acute episode. Occasionally, perivascular soft tissue thickening at the root of the celiac axis and SMA can also be seen in chronic pancreatitis and may mimic neoplastic vascular invasion [15] (Fig. 1a). This can be particularly challenging as there is an increased risk of developing pancreatic cancer in patients with chronic pancreatitis, developing in approximately 4% of chronic pancreatitis patients within 20 years [16]. Furthermore, the two entities share some imaging features, making differentiation of these diseases difficult [17]. In such cases, continued follow-up along with further evaluation with endoscopic ultrasound and fine needle aspiration for tissue diagnosis is warranted.

Perivascular soft tissue thickening and induration around the SMA and superior mesenteric vein are commonly recognized postoperative

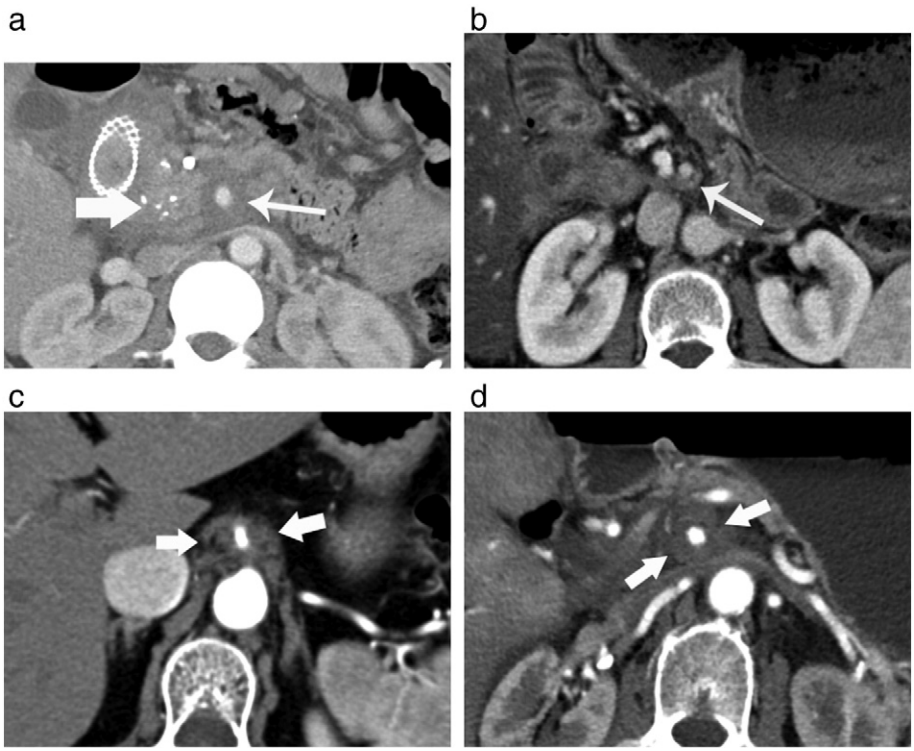

Fig. 1. Perivascular metastatic disease from pancreatic adenocarcnima and its mimickers. (a) A 4-year-old man with chronic calcific alcoholic pancreatitis. Axial contrast-enhanced CT image shows soft tissue thickening around the SMA (thin arrow) representing fibrosis that was stable on multiple follow-up examinations over the course of 3 years. Multiple foci of calcifications are seen in the pancreatic head (thick arrow) due to chronic pancreatitis. Endoscopic ultrasound with fine needle aspiration showed chronic inflammatory changes, and these changes remained stable over multiple follow-ups. (b) A 51-year-old man status post Whipple procedure for pancreatic adenocarcinoma. Axial contrast-enhanced CT image obtained 2 years postsurgery demonstrates small crescent-shaped soft tissue abutting the SMA (arrow) with no interval growth since surgery. This is a common finding after pancreaticoduodenectomy, and recurrence in this area should only be suspected in the presence of an increasing size and attenuation of the adjacent vessels. (c) A 68-year-old woman with vasculitis. Axial contrast-enhanced arterial phase CT image demonstrates soft tissue encasement of the celiac axis (thick arrows). The pancreas was normal in appearance without a mass lesion or ductal dilation. The perivascular soft tissue thickening completely resolved on the 6-month follow-up CT study. (d) A 77-year-old man with pancreatic adenocarcinoma. Axial contrast-enhanced arterial phase CT image demonstrates soft tissue encasement of the SMA (thick arrows) without an obvious mass lesion in the pancreas. Due to high clinical suspicion and elevated CA 19-9, metastatic pancreatic adenocarcinoma was confirmed on exploratory laparotomy sampling of the soft tissue.