

# Pancreatic Imaging

A Pattern-Based Approach  
to Radiologic Diagnosis  
with Pathologic Correlation

Atif Zaheer · Elliot K. Fishman  
Meredith E. Pittman · Ralph H. Hruban  
*Editors*

 Springer

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*To my parents for the inspiration,  
my wife for her undying support,  
my sons Asad and Ali for making it all worthwhile,  
and all pancreatologists, past, present, and future*

A.Z.

*To Max Saul Zember and my hope for his most  
promising future to help lead the next generation in  
discovery and wonder*

E.K.F.

*To all of my pathology teachers*

M.E.P.

*To the next generation of talented radiologists,  
including our dear Mila*

R.H.H.

---

## Preface

Case-based teaching has been a tradition in radiology education and continues to be a popular format for radiologists-in-training and practicing radiologists alike, as it mirrors radiology practice. A myriad of pathologies with distinctive imaging findings can be seen in the pancreas, and it is only natural to compile them based on their radiologic appearances. The integration of gross pathology into the teaching of radiology helps visualize the anatomic basis for radiologic changes. The pattern-based approach presented here allows the reader to use this book as a reference in their daily practice and to tackle cases based on their radiographic appearance on CT and MRI, to formulate a differential diagnosis, and to integrate the imaging findings with the gross pathology findings and the clinical context. Each case is presented as an unknown with a brief history, followed by a description of the imaging findings, differential diagnosis, discussion of salient points of the entity and its appearance on imaging, and ultimately a teaching point.

The entire spectrum of pancreatic pathology from anatomic variants to inflammation and to common, uncommon, and rare malignancies is covered in this book. This book is organized in two main sections comprising pancreatic parenchymal and ductal disease for easy navigation and reading. It is further subdivided into sections in which each entity is categorized based on enhancement pattern, solid or cystic nature, calcification, and focality.

The collection of cases in this book draws from the long experience gathered at Johns Hopkins over many years. Most of these cases are reminiscent of the lengthy discussions at the multidisciplinary conferences religiously held at Johns Hopkins. The cases ultimately provide the essence of the debates held between the radiologists, pathologists, gastroenterologists, oncologists, and pancreatic surgeons. We hope that we have been able to represent the expertise and passion of these physicians in the field of pancreatology, creating a valuable resource and an efficient reference for radiologists-in-training, practicing radiologists, and clinicians interested in imaging to provide the very best medical care.

Atif Zaheer  
On behalf of the co-editors and authors

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## **Part I**

### **Parenchymal Abnormalities: Focal Hypoenhancing Mass**

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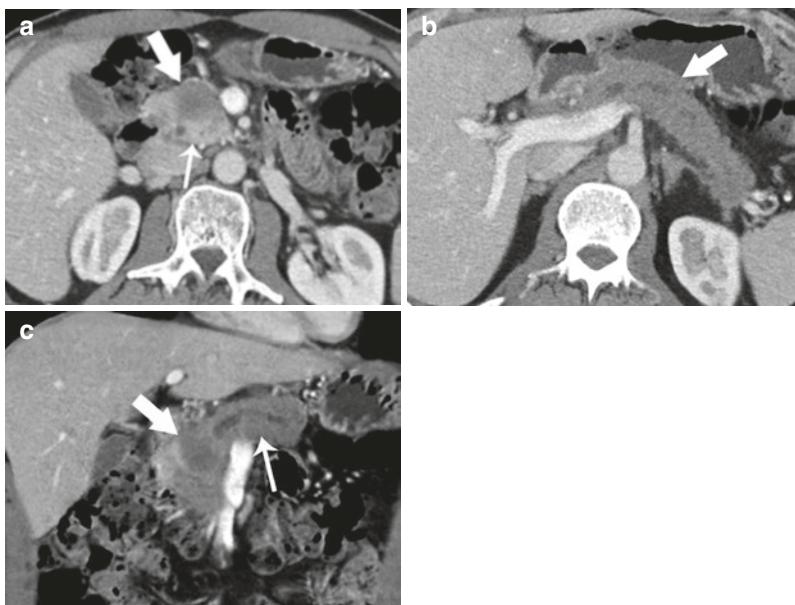
# Case 1: Pancreatic Adenocarcinoma with Upstream Textural Changes

Stephanie Coquia

---

## Clinical History

54-year-old man with epigastric pain. Family history was pertinent for mother with pancreatic adenocarcinoma.



**Fig. 1**

---

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## Imaging Findings

Axial post-contrast venous phase CT image demonstrates a hypoenhancing mass in the pancreatic head (Fig. 1a, *thick arrow*). Note the normal enhancement in the uncinate process of the pancreas (Fig. 1a, *thin arrow*). Diffuse hypoenhancement is seen in the pancreatic body and tail (Fig. 1b, *arrow*) with upstream dilation of the main pancreatic duct. A discrete mass is appreciated in the pancreatic head (Fig. 1c, *thick arrow*) which appears more mass-like compared to the diffuse hypoenhancement of the pancreatic tail (Fig. 1c, *thin arrow*).

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## Differential Diagnosis

Pancreatic adenocarcinoma and autoimmune pancreatitis.

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## Diagnosis

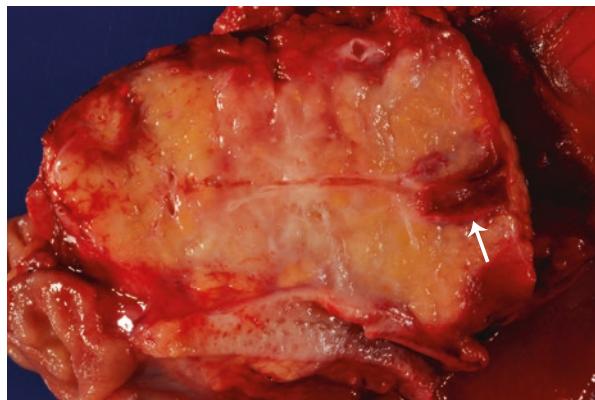
Pancreatic adenocarcinoma.

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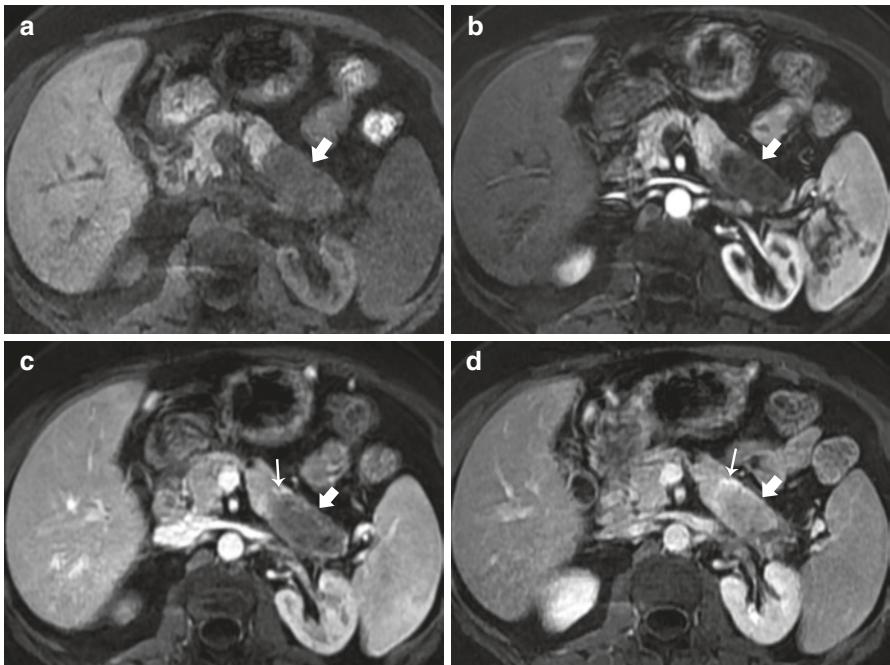
## Discussion

Most cases of pancreatic adenocarcinoma (PDAC) are sporadic; however, 5–10% of patients have a family history of the disease. There is an increased risk of developing PDAC in patients with a first-degree relative with pancreatic adenocarcinoma. Cancer susceptibility syndromes such as hereditary breast and ovarian cancer, hereditary pancreatitis, Peutz-Jeghers syndrome, Lynch syndrome and familial atypical multiple mole melanoma all increase the risk of developing pancreatic adenocarcinoma [1]. For instance, patients with Peutz-Jeghers syndrome have a 130-fold increased risk of developing pancreatic cancer.

Typical CT imaging appearance of PDAC is the presence of a hypodense and hypoenhancing mass with respect to the surrounding uninvolved pancreatic parenchyma and upstream dilation of the main pancreatic duct due to duct obstruction (Fig. 1). It is most conspicuous on the pancreatic parenchymal phase (approximately 40 s after the administration of contrast). PDAC is hypointense on T1-weighted pre-contrast fat-saturated images because of its fibrotic nature from the desmoplastic stromal response to the malignancy [2] (Fig. 3). For this reason, delayed and rim enhancement may also be appreciated on post-contrast imaging (Fig. 3). Textural changes in the uninvolved upstream pancreatic parenchyma (Fig. 1b, c) due to superimposed pancreatitis or fibrosis from long-standing obstruction may sometimes occur, and these can appear similar to the changes seen in autoimmune pancreatitis (AIP) [3]. Serum IgG4 may help evaluate the patient for AIP, but variable sensitivity has been reported in the literature ranging from 44% to 70%. Some of the features that are typical for AIP and may help



**Fig. 2** The patient illustrated in Fig. 1 underwent Whipple procedure. The surgical resection specimen shows a sclerotic, white, stellate mass impinging on the pancreatic duct with ductal dilation upstream from the mass (Fig. 2, arrow). Histologically, the mass was found to be a pancreatic adenocarcinoma.



**Fig. 3** Companion case. 58-year-old woman with PDAC. Note the presence of a hypointense mass on T1-weighted image (a, arrow) that demonstrates progressive enhancement on the arterial (b, arrow), venous (c, thick arrow) and delayed (d, thick arrow) phases after the administration of intravenous contrast. Note peripheral enhancement on the venous (c, thin arrow) and delayed phases (d, thin arrow)

differentiate AIP from PDAC include the presence of sausage-shaped appearance of the pancreas, delayed enhancement, hypodense capsule-like rim and wall thickening of the common bile duct [3, 4]. Patients with AIP can be treated with steroids rather than surgery, and imaging remains integral in differentiating PDAC from AIP [4, 5].

---

## Teaching Point

In patients with PDAC, textural changes in the uninolved upstream pancreatic parenchyma are due to superimposed pancreatitis or fibrosis from long-standing obstruction.

---

## References

1. Salo-Mullen EE, O'Reilly EM, Kelsen DP, Ashraf AM, Lowery MA, Yu KH, et al. Identification of germline genetic mutations in patients with pancreatic cancer. *Cancer*. 2015;121(24):4382–8. doi:[10.1002/cncr.29664](https://doi.org/10.1002/cncr.29664).
2. Hakime A, Giraud M, Vullierme MP, Vilgrain V. MR imaging of the pancreas. *J Radiol*. 2007;88(1 Pt 1):11–25.
3. Zaheer A, Singh VK, Akshintala VS, Kawamoto S, Tsai SD, Gage KL, et al. Differentiating autoimmune pancreatitis from pancreatic adenocarcinoma using dual-phase computed tomography. *J Comput Assist Tomogr*. 2014;38(1):146–52. doi:[10.1097/RCT.0b013e3182a9a431](https://doi.org/10.1097/RCT.0b013e3182a9a431).
4. Frulloni L, Scattolini C, Falconi M, Zamboni G, Capelli P, Manfredi R, et al. Autoimmune pancreatitis: differences between the focal and diffuse forms in 87 patients. *Am J Gastroenterol*. 2009;104(9):2288–94. doi:[10.1038/ajg.2009.327](https://doi.org/10.1038/ajg.2009.327).
5. Morselli-Labate AM, Pezzilli R. Usefulness of serum IgG4 in the diagnosis and follow up of autoimmune pancreatitis: a systematic literature review and meta-analysis. *J Gastroenterol Hepatol*. 2009;24(1):15–36. doi:[10.1111/j.1440-1746.2008.05676.x](https://doi.org/10.1111/j.1440-1746.2008.05676.x).

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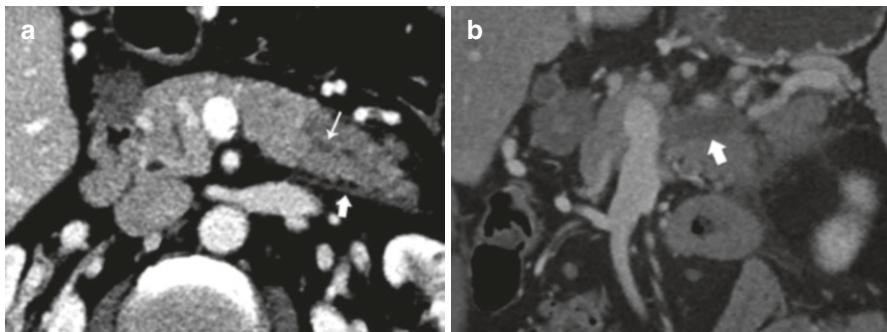
## Case 2: Pancreatic Adenocarcinoma with Superimposed Acute Pancreatitis

Stephanie Coquia

---

### Clinical History

66-year-old man presented with vomiting and mild mid-abdomen pain.



**Fig. 1**

---

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Johns Hopkins University, Baltimore, MD, USA  
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## Imaging Findings

Axial contrast-enhanced late arterial phase CT image demonstrates hypoattenuation of the pancreatic tail compared to the normal-appearing pancreatic head and body with associated peripancreatic stranding (Fig. 1a, *thick arrow*) and abrupt dilation of the main pancreatic duct (Fig. 1a, *thin arrow*). Coronal contrast-enhanced venous phase maximal intensity projection (MIP) CT image demonstrates occlusion of the splenic vein (Fig. 1b, *arrow*) with associated collaterals in the left upper quadrant.

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## Differential Diagnosis

Focal acute pancreatitis, pancreatic adenocarcinoma with superimposed pancreatitis, and autoimmune pancreatitis.

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## Diagnosis

Pancreatic adenocarcinoma with superimposed pancreatitis.

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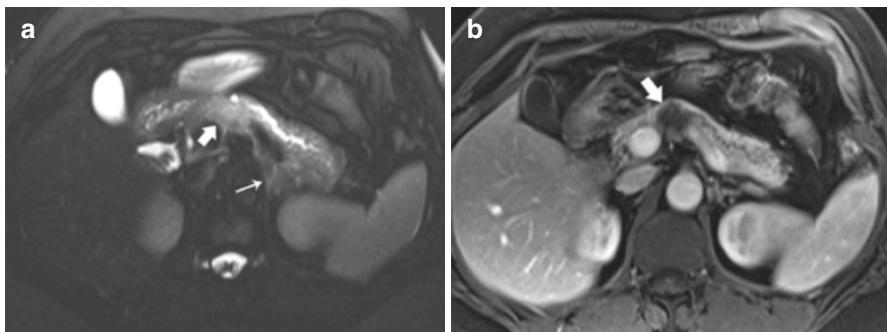
## Discussion

Pancreatic adenocarcinoma (PDAC) in rare instances may precipitate an episode of acute pancreatitis which may be secondary to mechanical obstruction of the pancreatic duct and ischemia caused by vascular invasion by the neoplastic cells or by direct activation of pancreatic enzymes [1]. In most cases patients present with a mild form of acute pancreatitis [1, 2].

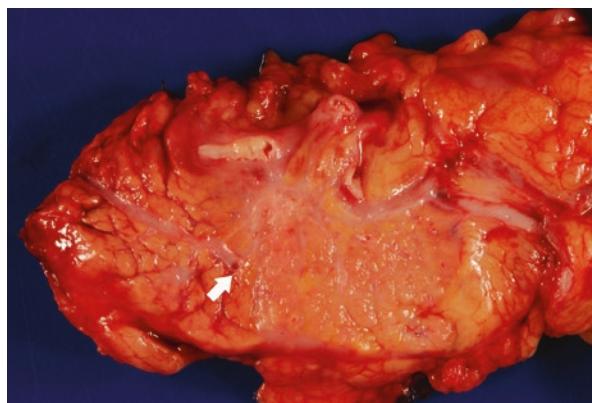
Acute pancreatitis may be the first presentation of PDAC (Figs. 1 and 2), and the diagnosis of an occult malignancy may be considered in patients above the age of 40 years who do not have obvious risk factors for pancreatitis, such as gallstones or alcohol abuse [3].

Abrupt dilation of the pancreatic duct within the region of abnormality is the key finding in this case and is indicative of an obstructing lesion (Figs. 1a and 2a). Autoimmune pancreatitis may also present as a geographic area of low attenuation, but pancreatic ductal dilation is usually absent [4]. Serum IgG4 may be a helpful adjunct as it can be elevated in patients with autoimmune pancreatitis. In this patient, splenic vein thrombosis is a nonspecific finding as it can be seen in 7–20% of patients with acute pancreatitis and does not necessarily favor the diagnosis of PDAC [5]. Follow-up CT or MR may be necessary after the acute episode of pancreatitis, when the inflammatory changes have resolved, in order to adequately evaluate the pancreas for an underlying mass. Alternatively, endoscopic ultrasound (EUS) may be performed to evaluate the presence of an occult mass.

The patient illustrated in Fig. 1 underwent distal pancreatectomy and pathology revealed PDAC (Fig. 3).



**Fig. 2** Companion case. 48-year-old woman with abdominal pain and elevated serum lipase. Axial T2-weighted image demonstrates a hyperintense mass in the pancreatic body (a, thick arrow) and upstream pancreatic ductal dilation. Peripancreatic inflammatory changes are present (a, thin arrow). Axial contrast-enhanced T1-weighted image demonstrates a hypoenhancing mass in the pancreatic body (b, arrow) with upstream pancreatic ductal dilation. Patient underwent distal pancreatectomy for PDAC



**Fig. 3** The surgical resection specimen showed a probe patent pancreatic duct at the pancreatic margin (left) with an abrupt blockage midway through the specimen. The pancreas was bivalved to show normal tan, lobulated pancreatic parenchyma at the margin with loss of the normal architecture at the area where the duct disappears (arrow). This firm area had a heterogeneous appearance with yellow fat/atrophy and white bands of fibrosis. The differential diagnosis included both PDAC and, less likely, chronic pancreatitis. Histologic sections showed an undifferentiated PDAC

---

### Teaching Point

Diagnosis of an occult malignancy may be considered in patients with otherwise no obvious risk factors for acute pancreatitis especially in the presence of an abruptly dilated pancreatic duct.

## References

1. Mujica VR, Barkin JS, Go VL. Acute pancreatitis secondary to pancreatic carcinoma. Study Group Participants. *Pancreas*. 2000;21(4):329–32.
2. Kohler H, Lankisch PG. Acute pancreatitis and hyperamylasaemia in pancreatic carcinoma. *Pancreas*. 1987;2(1):117–9.
3. Munigala S, Kanwal F, Xian H, Scherrell JF, Agarwal B. Increased risk of pancreatic adenocarcinoma after acute pancreatitis. *Clin Gastroenterol Hepatol*. 2014;12(7):1143–50. e1 doi:[10.1016/j.cgh.2013.12.033](https://doi.org/10.1016/j.cgh.2013.12.033).
4. Zaheer A, Singh VK, Akshintala VS, Kawamoto S, Tsai SD, Gage KL, et al. Differentiating autoimmune pancreatitis from pancreatic adenocarcinoma using dual-phase computed tomography. *J Comput Assist Tomogr*. 2014;38(1):146–52. doi:[10.1097/RCT.0b013e3182a9a431](https://doi.org/10.1097/RCT.0b013e3182a9a431).
5. Heider TR, Azeem S, Galanko JA, Behrns KE. The natural history of pancreatitis-induced splenic vein thrombosis. *Ann Surg*. 2004;239(6):876–80. discussion 80–2.

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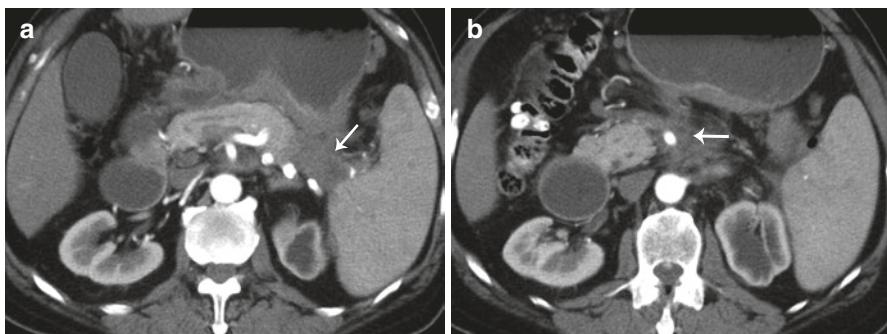
## Case 3: Pancreatic Adenocarcinoma, Unresectable

Stephanie Coquia

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### Clinical History

74-year-old man presented with early satiety, weight loss, postprandial nausea and vomiting, and intermittent abdominal pain.



**Fig. 1**

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## Imaging Findings

Axial contrast-enhanced arterial phase CT image demonstrates an ill-defined hypoattenuating mass (Fig. 1a, arrow) in the pancreatic tail extending to the splenic hilum. Axial contrast-enhanced arterial phase CT image demonstrates infiltrative soft tissue encasement of the superior mesenteric artery (SMA) (Fig. 1b, arrow).

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## Differential Diagnosis

Pancreatic adenocarcinoma and acute pancreatitis.

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## Diagnosis

Pancreatic adenocarcinoma, unresectable.

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## Discussion

Pancreatic adenocarcinoma frequently invades the periarterial neural plexus and lymphatics once it infiltrates the extrapancreatic fatty tissue. Radical resection of the superior mesenteric artery (SMA), a major supplier of blood to the gastrointestinal tract, has a high risk of bowel ischemia and death. In addition, even in the rare cases when this neurovascular tissue is resected, the patients are often left with positive margins that increase the risk of recurrent disease.

Tumor abutment of the SMA, defined as tumor contacting the artery for  $\leq 180^\circ$  of its circumference, may be considered borderline resectable disease. However, encasement ( $>180^\circ$ ) of the SMA precludes from resection [1, 2]. In the absence of encasement, SMA and the other major arteries in this region should be evaluated for the presence of variant anatomy especially related to the origin of the hepatic artery for presurgical planning as the presence of variant anatomy increases the risk for postoperative complications such as hemorrhage, pseudoaneurysm formation, and ischemia.

Tumor contact with the adjacent vessels can be well appreciated on CT and MRI. Tumor encasement of arteries ( $>180^\circ$ ) seen on CT has a sensitivity of up to 80% and a specificity of 98% for tumor invasion [3] (Fig. 1).

In the present case, the patient's slowly progressive symptoms and the presence of an infiltrative mass in the pancreas exclude acute pancreatitis as the diagnosis.

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## Teaching Point

Tumor encasement of the SMA precludes surgery as a treatment option.

## References

1. Evans DB, George B, Tsai S. Non-metastatic pancreatic cancer: resectable, borderline resectable, and locally advanced-definitions of increasing importance for the optimal delivery of multimodality therapy. *Ann Surg Oncol.* 2015;22(11):3409–13. doi:[10.1245/s10434-015-4649-2](https://doi.org/10.1245/s10434-015-4649-2).
2. Katz MH, Marsh R, Herman JM, Shi Q, Collison E, Venook AP, et al. Borderline resectable pancreatic cancer: need for standardization and methods for optimal clinical trial design. *Ann Surg Oncol.* 2013;20(8):2787–95. doi:[10.1245/s10434-013-2886-9](https://doi.org/10.1245/s10434-013-2886-9).
3. Lu DS, Reber HA, Krasny RM, Kadell BM, Sayre J. Local staging of pancreatic cancer: criteria for unresectability of major vessels as revealed by pancreatic-phase, thin-section helical CT. *AJR Am J Roentgenol.* 1997;168(6):1439–43. doi:[10.2214/ajr.168.6.9168704](https://doi.org/10.2214/ajr.168.6.9168704).