

Tumor-Vessel Relationships in Pancreatic Ductal Adenocarcinoma at Multidetector CT: Different Classification Systems and Their Influence on Treatment Planning¹

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Abbreviations: PDAC = pancreatic ductal adenocarcinoma, SMA = superior mesenteric artery, SMV = superior mesenteric vein

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Content Codes: **CT** **GI** **OI** **VA**

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See discussion on this article by Lillemoe (pp 113–115).

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe peripancreatic normal and variant vascular anatomic structures pertinent to the spread of pancreatic cancer and the different vascular procedures that can be performed for tumor resection.
- Identify the disease status of pancreatic cancer at multidetector CT with respect to resectable, borderline resectable, and unresectable disease.
- Describe key CT descriptors of venous and arterial involvement by pancreatic cancer to evaluate tumor resectability.

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Treatment of pancreatic ductal adenocarcinoma (PDAC) remains a challenge, given its propensity for early systemic spread and growth into the adjacent vital vascular structures. With the advent of newer surgical techniques and chemoradiation therapies, multidetector computed tomography (CT) plays a crucial role in the identification of patients with borderline resectable disease who may benefit from such treatments. Stage III PDAC is divided into two categories—*locally advanced*, defined by arterial encasement or nonreconstructible portovenous axis involvement; and *borderline resectable*, defined by limited arterial involvement and/or reconstructible portovenous involvement. A consensus definition for stage III borderline resectable PDAC has been proposed by the Americas Hepato-Pancreato-Biliary Association, the Society of Surgical Oncology, and the Society for Surgery of the Alimentary Tract and has gained widespread use. Evaluation of borderline resectable disease involves the identification of the circumferential and longitudinal relationship of the tumor with its neighboring vessels, markers of vascular invasion, and aberrant anatomic structures that alter the surgical approach. Furthermore, the use of template-based radiology reporting may increase the objectivity of the evaluation and mandate the provision of all of the key descriptors required for a comprehensive evaluation of the disease. In this review, the staging of PDAC at multidetector CT is described, with reference to the evaluation of the tumor-vessel interface as it guides treatment planning, along with a discussion of the key descriptors of PDAC at multidetector CT and their importance. Examples are provided of the imaging findings of borderline resectable disease and different surgical approaches, along with a discussion on the importance of standardized terminology and template-based reporting.

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Introduction

Pancreatic ductal adenocarcinoma (PDAC) is the fourth leading cause of cancer deaths in the United States, with an overall patient survival rate of 6%–7% (1). Multimodality treatment strategies, of which surgical resection is an integral part, remain the only option for cure. However, at the time of diagnosis of PDAC, less than 20% of the patients present with surgically resectable disease, and the remainder of the patients present with involvement of major abdominal vessels and/or distant metastatic disease (2). Tumor invasion of the superior mesenteric artery (SMA), superior mesenteric vein (SMV), celiac artery, and portal vein is common from PDAC arising in the pancreatic head because of their proximity, which makes

TEACHING POINTS

- Stage III borderline resectable tumor is characterized by a localized tumor abutting a major artery, including the celiac artery, common hepatic artery, or SMA. With regard to the portovenous axis, any degree of involvement falls into the category of borderline resectable disease as long as the vein can be technically resected and reconstructed.
- The change of the normal shape of the portal vein or SMV to a teardrop shape on axial multidetector CT images that is caused by tumor encasement or by tethering by adjacent fibrosis is referred to as the “teardrop sign” and is highly associated with vascular invasion.
- Careful scrutiny of the tumor-vessel interface and the length of involvement can be performed by supplementing the axial data with multiplanar reconstruction as well as volume rendering and maximum intensity projection techniques.
- The wall of the artery is thicker than that of the vein, and the flow rate in the artery is higher than that in the vein, so any change in the caliber of the artery or the presence of thrombus in the artery carries a higher risk of invasion than those findings in the vein.
- Template-based reporting should help decrease the incomplete documentation of descriptors that define the tumor-vessel relationship, and such incomplete documentation interferes with the decision-making process for patients with PDAC.

margin-negative resection challenging. Historically, vascular involvement was accepted as a sign of unresectability for PDAC. However, in studies since the early 1990s, investigators have shown that the survival of patients undergoing margin-negative venous resection is equivalent to that of patients undergoing standard pancreaticoduodenectomy and is superior to that for locally advanced disease treated without surgical resection (3–5). The development of newer vascular reconstruction techniques and the improvements in neoadjuvant therapies have now made disease with limited vascular involvement potentially resectable (6,7). Minimally invasive (laparoscopic and robotic) pancreaticoduodenectomy and distal pancreatectomy are performed safely and result in a shorter hospital stay, earlier recovery, and decreased delay in postoperative adjuvant therapy, with technical success similar to that for open surgery (8,9). Furthermore, the centralization of PDAC resection surgeries to high-volume centers has resulted in a decrease in the postoperative mortality to as low as 1%–3% (10).

Multidetector computed tomography (CT) is the most widely used and best validated imaging modality to assess local extension of the disease, perivascular disease, and distant metastasis, with accuracies of up to 77% for predicting resectability and 93% for predicting unresectability (11). The most common reason for abortion of surgery in patients whose cancer is otherwise considered resectable on the basis of the multidetector CT

findings is the presence of small liver metastases, followed by vascular encasement and peritoneal metastatic disease detected during surgery (11,12). In the absence of distant metastatic disease, the degree of vascular involvement at multidetector CT is considered the most important factor in the prediction of PDAC resectability and survival (13,14). Although conceptually simple, in practice there are nuances in differentiating arterial abutment from encasement and what constitutes reconstructible involvement of the portovenous axis. A multidisciplinary approach including close cooperation among surgeons, oncologists, radiation oncologists, pathologists, and radiologists is therefore required to improve survival in patients with resectable disease and in those with borderline resectable disease. Furthermore, standardized template-based radiology reporting is recommended to create uniformity in the evaluation of the disease among physicians and across institutions, to ensure that all of the key descriptors are addressed in the imaging evaluation of PDAC.

For the purpose of standardized care, it is imperative for radiologists to know the key descriptors and understand the role that they play in guiding surgical and oncologic treatment. In this article, the key multidetector CT descriptors that determine tumor resectability are described with examples, and strategies for treatment planning are outlined, as well as the importance of standardization of radiology reporting for evaluation of PDAC.

Imaging Technique

Multidetector CT protocols for pancreatic imaging may vary at different institutions but typically include a multiphase technique with thin-section imaging and multiplanar reconstruction. Contrast material-enhanced images usually include a late arterial or pancreatic phase and a portal venous phase after administration of intravenous contrast material consisting of 100–120 mL of iodinated contrast material (iodixanol injection [Visipaque 320; GE Healthcare, Waukesha, Wis] or iohexol injection [Omnipaque 350; GE Healthcare]), usually at an injection rate of 4–5 mL/sec. The late arterial or pancreatic phase is acquired at 35–50 seconds for optimum evaluation of the pancreatic parenchyma and for the assessment of peripancreatic arterial anatomic structures. The portal venous phase, which is acquired at 60–90 seconds, allows for optimum assessment of the venous anatomic structures and their involvement, along with detection of hepatic and distant metastatic disease (15). Water can be used as a negative oral contrast agent. Multidetector

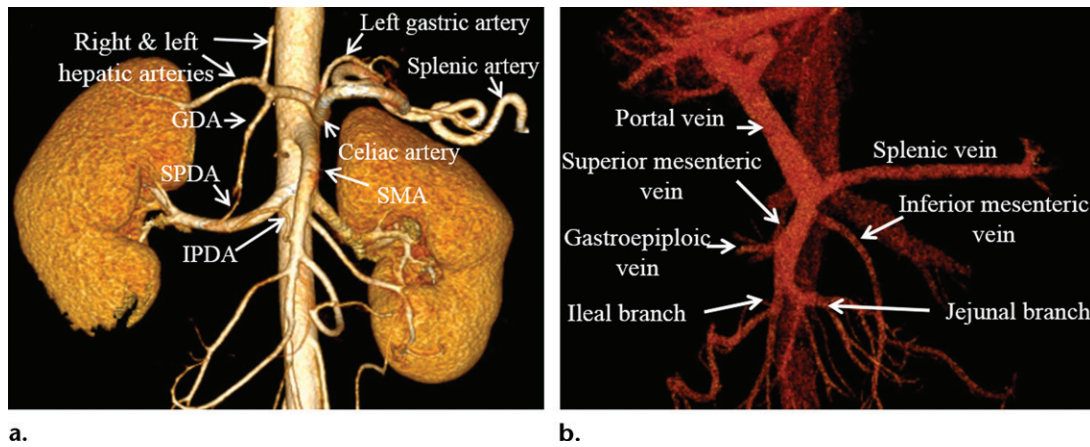


Figure 1. Peripancreatic vascular anatomic structures. Volume-rendered CT images show normal peripancreatic arterial (a) and venous (b) anatomic structures with images obtained in the arterial and venous phases, respectively. *GDA* = gastroduodenal artery, *IPDA* = inferior pancreaticoduodenal artery, *SPDA* = superior pancreaticoduodenal artery.

CT may include both thick (3–5-mm) sections for primary review and thin (0.75–1.50-mm) sections for image postprocessing. Postprocessed images include multiplanar reformatted images (coronal and sagittal), maximum intensity projection images, and volume-rendered images, which are valuable in the identification of subtle changes in vascular calibers. Volume-rendered images may help assess vessels in the optimal plane for variant anatomic structures, tumoral involvement, and collateral pathways (16). Knowledge of normal and variant anatomic structures is imperative to assess perivascular disease and to guide surgery for possible vascular reconstruction (Fig 1).

Although multidetector CT is most widely used, magnetic resonance (MR) imaging is an excellent modality for the workup of pancreatic carcinoma. On the basis of the limited available data, it has been suggested that MR imaging and CT are equivalent for assessment of vascular invasion (17) and have similar performance in the preoperative evaluation of PDAC (18). Compared with multidetector CT, MR imaging has demonstrated significantly greater tumor conspicuity of small PDACs ($P < .05$), which may be isoattenuating or slightly hypoattenuating on multidetector CT images (19). This conspicuity may better delineate the interface of such tumors with the adjacent vessels. MR imaging has also been shown to allow improved detection of metastatic disease, especially in the liver, with a sensitivity of up to 100%, compared with a CT sensitivity of up to 80%, for detection of hepatic metastases (20). Although multidetector CT is a preferred modality for PDAC staging at multiple institutions because of its wider availability, its lower cost compared with MR imaging, and institutional and patient preferences, MR imaging is recognized by the National Comprehensive Cancer Network as an important

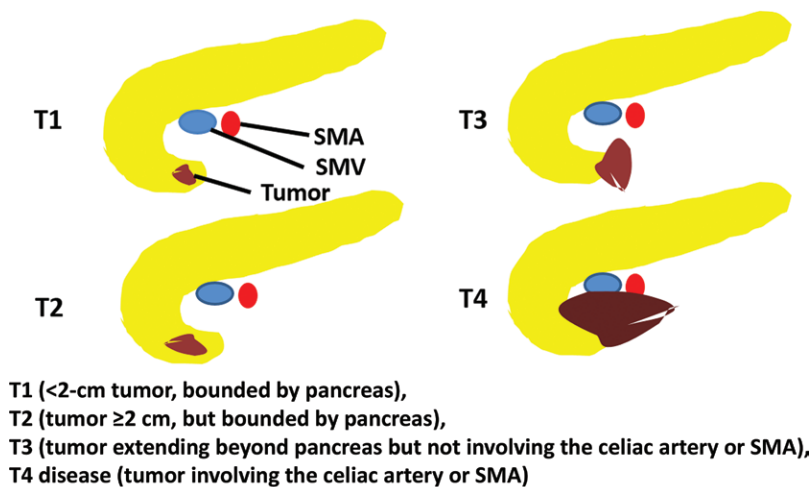
viable imaging option, particularly as an adjunct to CT in high-risk patients in whom CT findings are negative for metastatic disease or in patients with small or indeterminate hepatic lesions depicted at CT (21).

Preoperative dual-modality imaging with fluorine 18 (^{18}F) fluorodeoxyglucose positron emission tomography (PET) combined with CT (PET/CT) may serve as a predictor of survival in patients undergoing curative resection. Parameters such as the maximum standardized uptake value, the metabolic tumor volume, and the total lesion glycolysis obtained from the preoperative PET/CT examination are associated with recurrence-free survival and overall survival (22,23). However, these techniques may have a limited role in characterizing small lesions, and larger prospective studies are required to further elucidate the role of PET/CT.

Assessment of Resectability

PDAC Staging

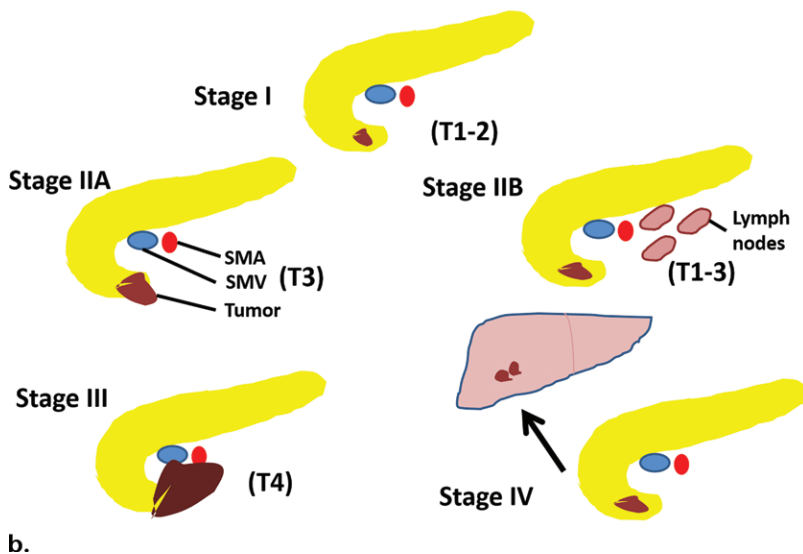
For PDAC, the staging system of the American Joint Committee on Cancer is most commonly used to assess the status of the tumor (T), lymph nodes (N), and metastatic disease (M) (24) (Fig 2). Preoperative imaging is used to characterize resectable, borderline resectable, locally advanced, and metastatic disease. Stages I and II are classified as clearly resectable, with the absence of tumor contact with the adjacent celiac trunk, hepatic artery, SMA, SMV, and portal vein. Stage IV is defined by the presence of distant metastatic disease and precludes resection because patients will derive no benefit from the operation. Patients with borderline resectable disease have received considerable attention with the advent of preoperative chemoradiation therapies and newer vascular reconstruction techniques. Stage III is defined as a



a.

Figure 2. Tumor grading and TNM staging of PDAC. (a) Drawing shows that PDAC is graded on the basis of the tumor size and involvement of the peripancreatic vasculature. The nodal staging includes the absence (N0) or presence (N1) of disease in regional nodes. M0 is defined as absence of metastatic disease, and M1 is the presence of distant metastatic disease. (b) Drawing shows staging of PDAC. Stage I is disease with the primary tumor confined to the pancreas without lymphadenopathy or metastatic disease. Stage IIA is disease extending beyond the pancreas without vascular involvement, lymphadenopathy, or metastatic disease. Stage IIB is disease with lymphadenopathy. Stage III is disease with vascular involvement. Stage IV is disease with distant metastatic disease.

b.



localized tumor with major vessel involvement and is further subdivided into two categories—locally advanced unresectable and borderline resectable. Stage III borderline resectable tumor is characterized by a localized tumor abutting a major artery, including the celiac artery, common hepatic artery, or SMA. With regard to the portovenous axis, any degree of involvement falls into the category of borderline resectable disease as long as the vein can be technically resected and reconstructed. In contrast, the extent of vessel involvement with locally advanced tumor is more extensive and by definition is not amenable to resection. This category of locally advanced unresectable disease includes encasement of one or more major arteries and/or involvement of the portovenous axis that is not technically reconstructible. Although the distinction between borderline resectable and locally advanced disease is conceptually simple, the precise definition has been variable and may be based on the imaging or clinical criteria. As a result of the imprecision in defining borderline

resectable and locally advanced disease, different classification systems exist. These systems include the National Comprehensive Cancer Network system, the MD Anderson system, the Intergroup system, and the most commonly used system, the Americas Hepato-Pancreato-Biliary Association/Society of Surgical Oncology/Society for Surgery of the Alimentary Tract (AHPBA/SSO/SSAT) system (25). As a result of the subjective nature of these definitions, the goal of the current review is to define borderline resectable PDAC by using a generalizable objective method that is based on the multidetector CT of each individual vessel in relation to its interface with the adjacent tumor and the surgical options for treatment in each of these cases.

Portal Vein and Superior Mesenteric Vein

Portal vein and/or SMV involvement is frequently seen in the tumors involving the head, neck, and proximal body of the pancreas. In general, venous

Table 1: Different Criteria for Venous Resectability of PDAC

Criteria	SMV and Portal Vein Involvement
National Comprehensive Cancer Network criteria	Tumor encasement and/or reconstructible occlusion
MD Anderson criteria	Occlusion
AHPBA/SSO/SSAT criteria	Occlusion
Intergroup criteria	Tumor encasement and/or reconstructible occlusion

Note.—AHPBA/SSO/SSAT = Americas Hepato-Pancreato-Biliary Association/Society of Surgical Oncology/Society for Surgery of the Alimentary Tract.

involvement is not an absolute contraindication for resection of PDAC (Table 1). The median length of survival in cases that require venous resection varies from 13 to 22 months, which is comparable to that in cases without a need for venous resection (26–28). Also, the 30-day postoperative mortality rate for patients undergoing pancreaticoduodenectomy with venous resection is 3.9%, compared with 3% for patients without venous resection (29). These results have encouraged surgeons to perform extended pancreatectomy with venous resection more frequently (30). Intimal invasion of the portal vein and SMV may result in a poorer survival, compared with that of patients without true vascular invasion (31). However, because of the severe desmoplastic reaction induced by PDAC, it may be difficult to differentiate adhesions from true invasion, and more than 20% of cases that show venous involvement at preoperative imaging do not have true venous invasion (31). Therefore, several attempts have been made to find predictors, such as the tumor-vein circumferential interface, the vein caliber, and the length of the tumor-vein interface at multidetector CT, to evaluate the possibility of tumor resection and venous invasion.

Tumor-Vein Circumferential Interface

In 1997, Lu et al (32) prospectively included 25 patients with PDAC who underwent local dissection for curative resection or palliative surgery and reported a grading system that used preoperative thin-section helical CT for grading the vascular (both arterial and venous) involvement with PDAC on the basis of the tumor-vessel circumferential contiguity. Lu et al (32) concluded that contiguity that exceeds 50% of the circumference of the vessel ($>180^\circ$) has a sensitivity of 84% and a specificity of 98% in predicting the probability of the need for vessel resection. Similarly, Loyer et al (33) showed that venous resection was not required in most patients when the tumor was separated from the vessels by a fat plane or by normal pancreatic parenchyma at CT. Loyer et al

(33) also found that tumor making contact with the vessel without a change in the caliber of the vessel lumen at CT was an unreliable predictor of tumor adherence to the vessel, which could be predicted reliably only when CT showed complete encircling of the tumor around the vessel. Also, a negative margin could not be obtained when the tumor completely encircled the vein or in the presence of vascular occlusion (33).

Other investigators have further verified that the extent of the tumor-vessel interface correlates with the probability of vascular invasion and can be used to predict (a) the need for vein resection and (b) survival (14,34). The change of the normal shape of the portal vein or SMV to a teardrop shape on axial multidetector CT images that is caused by tumor encasement or by tethering by adjacent fibrosis is referred to as the “teardrop sign” and is highly associated with vascular invasion (35) (Fig 3). At preoperative multidetector CT, the probability of vascular invasion is up to 40% for tumor abutment ($\leq 180^\circ$ contact), compared with 80% in the presence of tumor encasement ($>180^\circ$ contact), and 100% if the tumor is completely surrounding the portal vein or SMV (14,36,37).

Vein Caliber and Length of Interface

The Ishikawa classification was one of the first to describe the degree of venous involvement according to the change in caliber of the portal vein and/or SMV and the length of this change (13) (Figs 3, 4). Ishikawa et al (13) concluded that patients with normal calibers of the portal vein and the SMV, unilateral narrowing, and 1.2 cm or less (in length) of longitudinal involvement had more favorable long-term outcomes, compared with patients with bilateral venous narrowing, with or without collateral vein formation. The length of the venous resection also correlates with the long-term survival because the longer interposition grafts used for venous reconstruction are more prone to narrowing by external compression from postoperative fluid collections (38).

Figure 3. Imaging examples of the Ishikawa classification system. *PV* = portal vein, *T* = tumor. (a) Coronal contrast-enhanced CT image shows venous smooth shift without a change in the venous caliber. (b) Coronal contrast-enhanced CT image shows unilateral narrowing. (c) Coronal contrast-enhanced CT image shows circumferential narrowing of the portal-splenic venous confluence. Inset: Axial image shows the “teardrop” deformity (arrow) for bilateral narrowing of the portal-splenic venous confluence. (d) Coronal contrast-enhanced CT image shows circumferential narrowing of the portal-splenic venous confluence with development of collateral circulation (arrows).

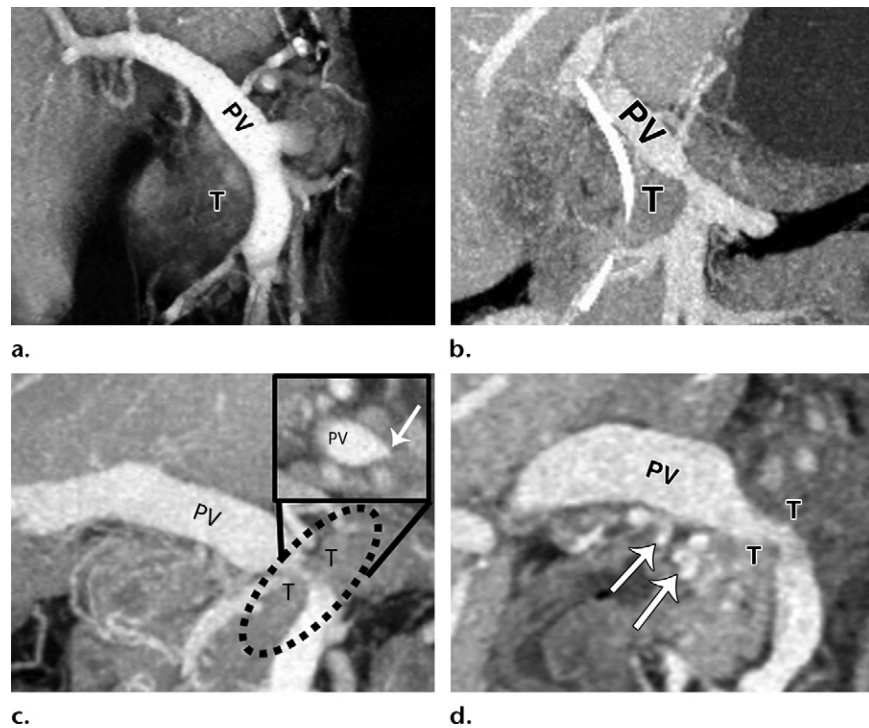
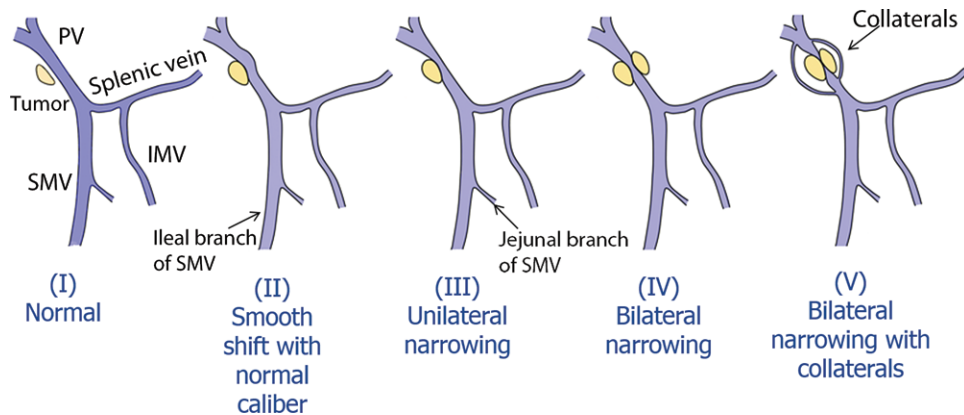


Figure 4. Ishikawa classification system. Diagrams show the Ishikawa system, one of the definition systems that describes the relationship between the pancreatic tumor and the nearby vessels, as well as the surgical outcomes of the resection. *IMV* = inferior mesenteric vein, *PV* = portal vein.



Venous caliber change at imaging was correlated with venous wall invasion in a study by Nakao et al (27) of 671 patients who underwent pancreatectomy and portal vein resection for PDAC. Nakao et al (27) demonstrated an absence of venous invasion at pathologic examination in the patients without a radiographic appearance of vascular involvement; and vascular invasion was seen at pathologic examination in 51%, 74%, and 93% of patients with radiographic unilateral narrowing of the portal vein, bilateral narrowing of the portal vein, and complete portal vein obstruction with collateral vein formation, respectively. Furthermore, long-term survival (>5 years) was observed in patients with an absence of venous involvement or with only unilateral narrowing observed at imaging (27).

Careful scrutiny of the tumor-vessel interface and the length of involvement can be performed

by supplementing the axial data with multiplanar reconstruction as well as volume rendering and maximum intensity projection techniques. The portal vein and SMV should be evaluated in their entirety in relation to the tumor on the coronal or sagittal images. Furthermore, the portal vein and SMV and their tributaries should also be evaluated on multiplanar reformatted images to assess the craniocaudal extent of the tumor.

Methods of Venous Reconstruction

The primary goal of venous reconstruction is to reconstitute flow after removal of the involved portal vein and/or SMV. It is not possible to anastomose a single venous lumen with multiple lumina; and therefore, a single and patent segment of the SMV below and a single patent segment of the portal vein or SMV above the involved venous segment are usually required

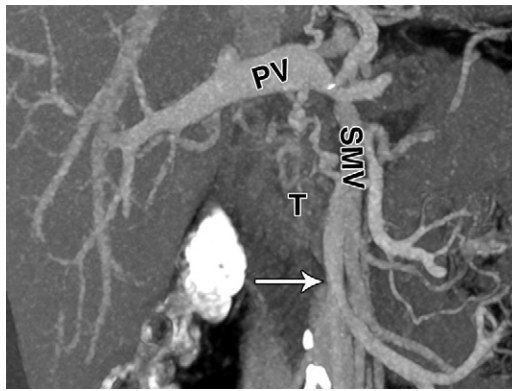


Figure 5. PDAC in the head of the pancreas in a 60-year-old woman. Coronal contrast-enhanced maximum intensity projection image shows the tumor (*T*), with circumferential narrowing of the portal vein (*PV*) and abutment of the SMV beyond the level of the trifurcation (arrow). Resection was not performed because of the difficulty of resection and reconstruction of the portal vein.

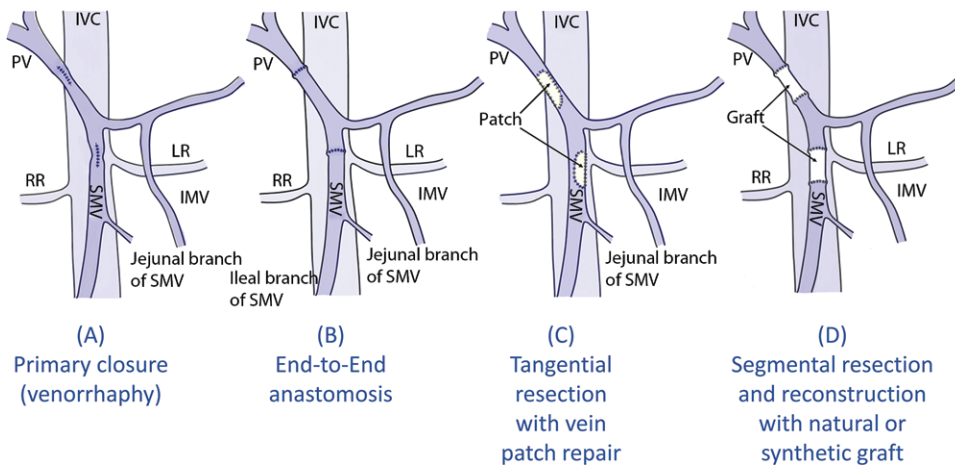


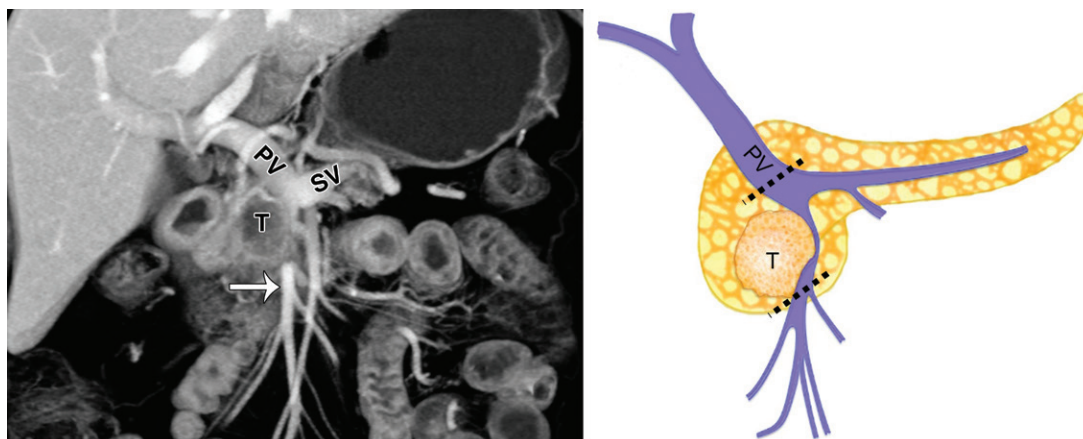
Figure 6. Diagrams of different methods of venous reconstruction: *A*, primary closure (venorrhaphy); *B*, end-to-end anastomosis; *C*, tangential resection with vein patch repair; and *D*, segmental resection and reconstruction with natural or synthetic graft. *IMV* = inferior mesenteric vein, *IVC* = inferior vena cava, *LR* = left renal vein, *PV* = portal vein, *RR* = right renal vein.

for successful venous reconstruction (Fig 5). Multiple surgical techniques have been used for venous reconstruction, such as primary-closure venorrhaphy, end-to-end anastomosis, tangential resection with patch repair, and reconstruction with a natural or synthetic graft (Fig 6). Tangential resection is performed in cases of venous wall involvement of less than 120° with venotomy closure primarily or with a saphenous vein graft to avoid venous narrowing. End-to-end tension-free anastomosis can be performed after resection of up to 4 cm of the length of the portal vein and/or SMV (Fig 7). An autologous venous interposition graft may be used if end-to-end anastomosis cannot be performed, and the most commonly used grafts are the internal jugular vein (39) (Fig 8a, 8b), the external iliac vein (40), the left renal vein (41), and the superficial femoral veins (42). A polytetrafluoroethylene interposition graft is another option for venous reconstruction, with minimal risk of hepatic necrosis and graft infection (39) (Fig 8c, 8d).

Isolated involvement of one of the first-order jejunal or ileal branches of the SMV can be treated with ligation, without the need for reconstruction, in the presence of preserved mesenteric flow through the remaining first-order branches

(Fig 9). In the presence of both jejunal and ileal first-order branch involvement, ligation of the jejunal branch along with segmental resection and reconstruction of the ileal branch with an interposition graft may be performed (43).

Another important consideration is the involvement of the portosplenomesenteric venous confluence by the tumor. In the case of involvement, the splenic vein can be ligated during surgery to facilitate venous reconstruction of the portal vein–SMV confluence. However, because the short gastric veins and the left gastroepiploic vein drain into the splenic vein, the venous flow from the spleen and the stomach is decreased, a finding that predisposes to left-sided portal hypertension and the formation of gastroesophageal varices that may bleed (Fig 10). One option is the reimplantation of the splenic vein into the reconstructed portal vein–SMV confluence after tumor resection (44). The other option may be determined by evaluating the anatomic configuration of the inferior mesenteric vein. In cases in which the inferior mesenteric vein drains into the splenic vein, the splenic vein allows retrograde flow from the stomach and the spleen to reach the portomesenteric circulation through the inferior mesenteric vein (Fig 11a). In cases in which

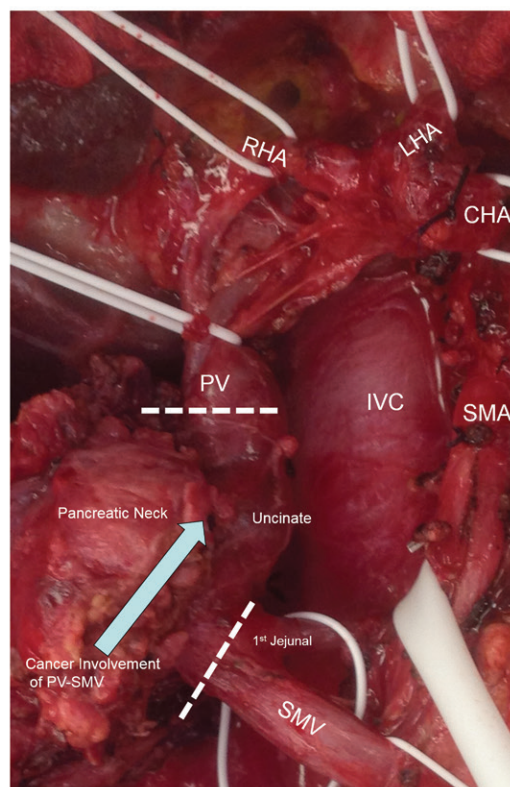


a. **Figure 7.** PDAC in the pancreatic head in a 56-year-old woman. PV = portal vein. **(a)** Coronal contrast-enhanced maximum intensity projection image shows tumor (T) arising in the pancreatic head, with unilateral narrowing of the portal vein and the SMV at the portosplenic junction, with an adequate uninvolved length of the distal SMV (arrow) for venous reconstruction. SV = splenic vein. **(b)** Drawing shows the tumor (T) in the pancreatic head; dotted lines = sites of resection of the portal vein and SMV. **(c)** Intraoperative photograph shows cancer involvement (arrow) of the portal vein and SMV. Both veins were resected (dashed lines) above and below the tumor at a level above the first jejunal branch. The length was adequate for a primary anastomosis because the splenic vein was divided and the mismatch between the diameters of the portal vein and SMV was not pronounced. CHA = common hepatic artery, IVC = inferior vena cava, LHA = left hepatic artery, RHA = right hepatic artery, SMA = superior mesenteric artery.

the inferior mesenteric vein drains directly into the SMV, a distal splenorenal shunt may be created to allow decompression of the splenic vein after its ligation (45) (Fig 11b).

Arterial Involvement

Sixty percent of cases of PDAC arise in the pancreatic head region (46), which predisposes the celiac artery, the hepatic artery, and the SMA to tumor involvement. Similarly, accessory or replaced right hepatic arteries are also at a higher risk of involvement by tumors of the pancreatic head because of the usual course of these arteries lateral and posterior to the bile duct and their frequent trajectory through the pancreatic head itself. In several studies, investigators have assessed the survival of patients with arterial resection. Although some groups have suggested similar morbidity and mortality in patients undergoing pancreaticoduodenectomy alone and those undergoing pancreaticoduodenectomy with arterial resection (39,47), a recent meta-analysis by Mollberg et al (48) included 26 retrospective studies comparing patients who underwent pancreatectomy with and without arterial resection. Mollberg et al (48) concluded that pancreatectomy with arterial resection is associated with higher



c.

rates of postoperative complications and a shorter overall survival than conventional pancreatectomy without arterial resection. SMA resection is usually not performed because of the high risk of bowel ischemia and death. However, with improved surgical techniques and the advancement of neoadjuvant and adjuvant therapies, arterial resection of other vessels is recommended in highly selected patients to obtain an R0 resection (margin negative) because of the potential survival benefit when compared with palliation alone (5,39). Such highly selected patients include younger patients with good performance status (such as Eastern Cooperative Oncology Group [ECOG] score), less-aggressive tumor biology,

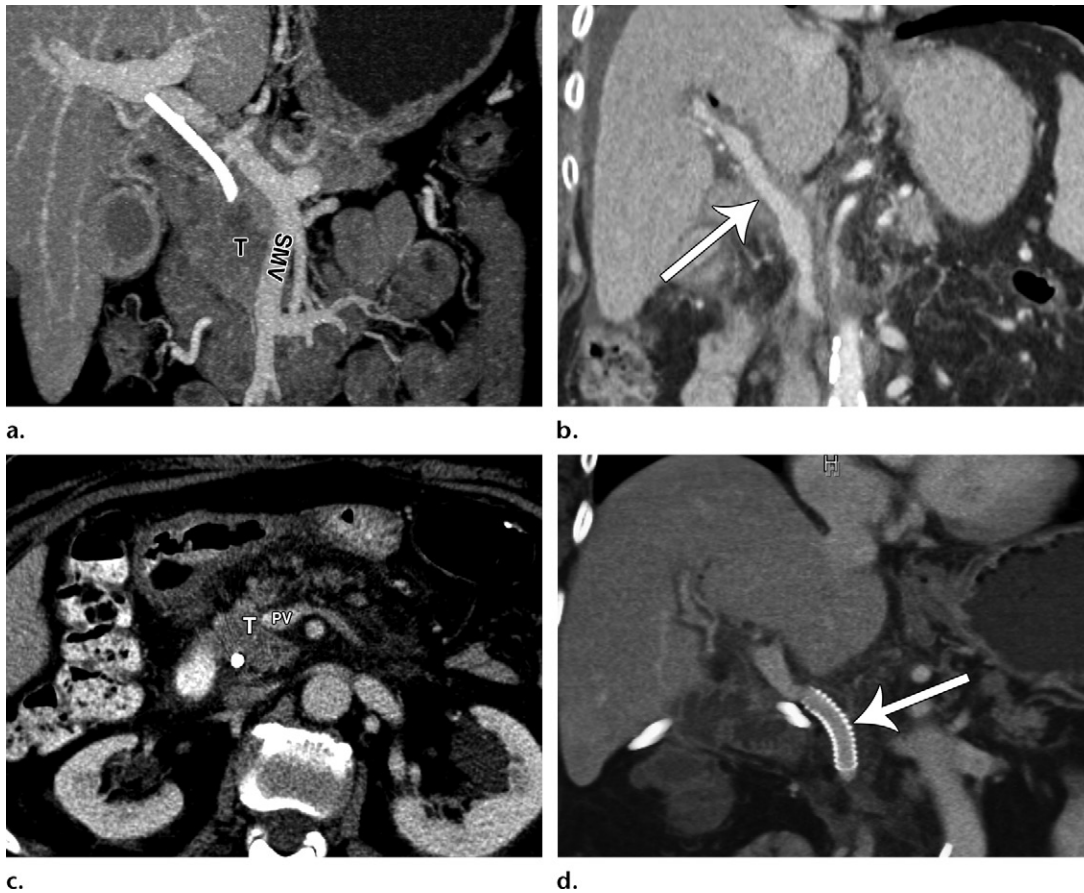


Figure 8. Venous reconstruction in two patients. (a, b) PDAC arising from the head of the pancreas in a 60-year-old woman. (a) Coronal maximum intensity projection CT image shows abutment of the SMV by the pancreatic head tumor (T), without luminal narrowing. (b) Coronal contrast-enhanced CT image obtained 1 month after the surgery shows a patent internal jugular vein graft (arrow) after Whipple surgery with portal vein resection. (c, d) PDAC in an 82-year-old woman. (c) Axial contrast-enhanced CT image shows abutment of the tumor (T) and the portal vein (PV). The patient underwent Whipple surgery with resection and reconstruction of the portal vein with a synthetic polytetrafluoroethylene graft. (d) Postoperative volume-rendered CT image obtained 1 week after surgery shows patency of the graft (arrow).

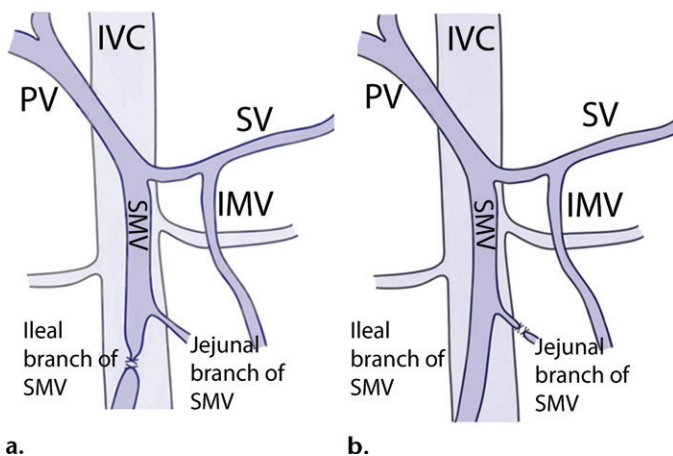


Figure 9. Diagrams of SMV branch ligation. Isolated involvement of one of the first-order ileal (a) or jejunal (b) branches of the SMV with tumor at pancreaticoduodenectomy can be treated with ligation of this branch without the need for reconstruction as long as the other branch is not involved. IMV = inferior mesenteric vein, IVC = inferior vena cava, PV = portal vein, SV = splenic vein.

and preoperative imaging findings suggestive of the feasibility of resection and reconstruction.

Criteria for Resectability

Similar to venous involvement, tumor encasement of arteries seen at multidetector CT has

a sensitivity of up to 80% and a specificity of 98% for invasion, but tumor abutment is not considered a sensitive sign for vascular invasion (Fig 12) (32). However, the arteries are evaluated in a slightly different fashion in the presence of arterial attenuation. The wall of the artery is



Figure 10. Recurrent hematemesis in a 51-year-old man 4 years after he underwent a Whipple procedure for PDAC. Axial contrast-enhanced CT image shows the development of massive collateral vessels (arrows) after splenic vein ligation.

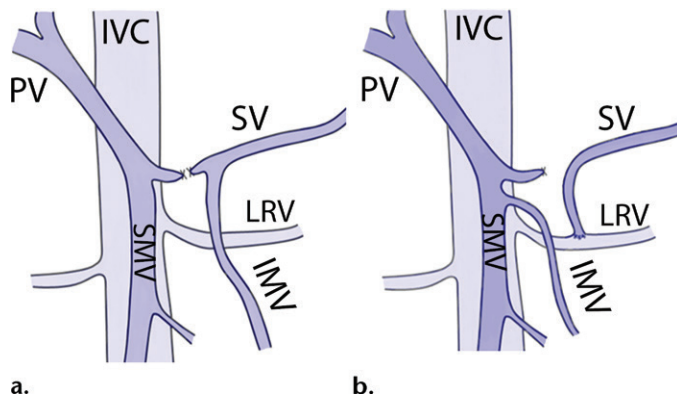


Figure 11. Diagrams of two types of splenic vein ligation that are based on the anatomic configuration of the inferior mesenteric vein. *IMV* = inferior mesenteric vein, *IVC* = inferior vena cava, *LRV* = left renal vein, *PV* = portal vein, *SV* = splenic vein. **(a)** The splenic vein may be ligated distal to the inferior mesenteric vein to allow retrograde venous flow to reach the portomesenteric circulation. **(b)** A splenorenal shunt may be created in the presence of direct drainage of the inferior mesenteric vein into the SMV.

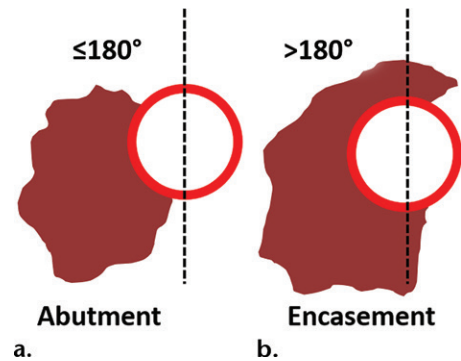


Figure 12. Diagrams of two types of arterial involvement with PDAC. **(a)** *Abutment* is defined as 180° or less of circumferential contact of the tumor with the vessel. **(b)** *Encasement* is defined as more than 180° of circumferential contact of the tumor with the vessel.

thicker than that of the vein, and the flow rate in the artery is higher than that in the vein, so any change in the caliber of the artery or the presence of thrombus in the artery carries a higher risk of invasion than those findings in the vein (49). The exact definition of borderline resectability is variable among institutions and is dependent on the surgical skills and the institutional experience (50). Variability exists in the resection criteria for celiac artery involvement, ranging from no involvement to abutment. In general, abutment of the common hepatic artery and SMA is considered borderline resectable disease, as summarized in Table 2. The pancreaticoduodenal artery is routinely resected as part of the Whipple procedure, and its isolated involvement does not affect the resectability status. Individual vessels are discussed in detail in the following sections.

Celiac and Hepatic Artery Involvement

Hepatic artery involvement results from the growth of PDAC cephalad in the head and neck of the pancreas. Therefore, in some cases, only a

short segment of the hepatic artery becomes involved at the origin of the gastroduodenal artery and is amenable to resection and reconstruction, with or without an interposition graft, because of the hepatic artery redundancy (51). Involvement of the celiac artery is one of the major criteria that define locally advanced PDAC unless deemed resectable and reconstructible (52,53). PDAC arising from the pancreatic neck is more likely to involve the celiac artery, along with involvement of a segment of the hepatic artery proximal to the origin of the gastroduodenal artery. This segment can be resected with distal pancreatectomy and splenectomy (modified Appleby procedure), without the need for reconstruction, in the presence of an uninvolved and patent gastroduodenal artery that restores blood supply to the liver by providing a collateral pathway between the gastroduodenal artery and the SMA via the pancreaticoduodenal artery (Fig 13). Therefore, it is important to evaluate (a) the extent of the tumor on the common hepatic artery proximal to the origin of the gastroduo-

Table 2: Different Criteria for Arterial Resectability of PDAC

Criteria	Celiac Artery	Common Hepatic Artery	SMA
National Comprehensive Cancer Network criteria	Head tumor: no abutment; body or tail tumors: abutment	Abutment or short-segment encasement allowing reconstruction	Abutment
MD Anderson criteria	Abutment	Abutment or short-segment encasement allowing reconstruction	Abutment
AHPBA/SSO/SSAT criteria	No abutment or encasement	Abutment or short-segment encasement allowing reconstruction	Abutment
Intergroup criteria	Abutment	Abutment or short-segment encasement allowing reconstruction	Abutment

Note.—AHPBA/SSO/SSAT = Americas Hepato-Pancreato-Biliary Association/Society of Surgical Oncology/Society for Surgery of the Alimentary Tract.

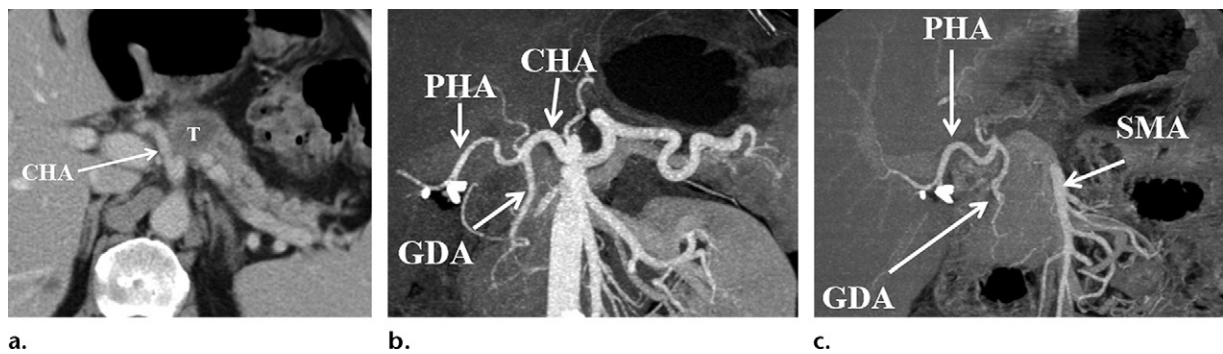


Figure 13. PDAC in the pancreatic body in a 59-year-old man. (a) Axial CT image shows a pancreatic body tumor (*T*) abutting the common hepatic artery (*CHA*). (b) Preoperative three-dimensional maximum intensity projection CT image shows a patent gastroduodenal artery (*GDA*). *CHA* = common hepatic artery, *PHA* = proper hepatic artery. (c) Postoperative three-dimensional maximum intensity projection CT image shows a successful common hepatic artery resection without the need for reconstruction, because the blood flow to the liver is maintained owing to the collateral pathway between the SMA and the proper hepatic artery (*PHA*) through the gastroduodenal artery (*GDA*).

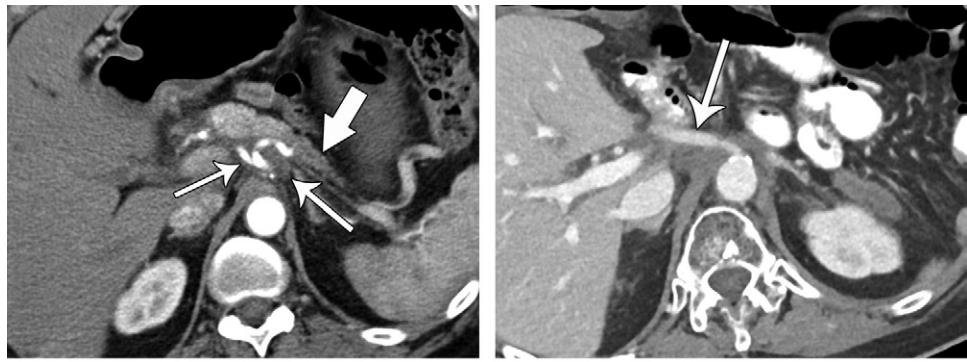
denal artery, (b) involvement of the gastroduodenal artery or extent to the hepatic hilum, and (c) the collateral pathway between the SMA and the gastroduodenal artery (54). Blood flow in the proper hepatic artery after occlusion of the common hepatic artery is confirmed during surgery by assessing the presence of pulsations or arterial Doppler signal, to avoid hepatic ischemia. Arterial reconstruction may be considered to increase flow to the liver (Fig 14).

Gastric ischemia is another frequent complication caused by disruption of flow to the left gastric and left gastroepiploic arteries (Fig 15) (54). Ischemic complications may be avoided by preoperative coil embolization of the common hepatic artery and celiac artery to promote development of collateral vessels to the liver via the pancreaticoduodenal arcade and to the stomach via the right gastric, right gastroepiploic, and left phrenic arteries (55). PDAC extending to the right and the left hepatic arteries requires complex vascular reconstruction by using autologous transposition arterial grafts (56) or a bifurcated

jump graft from the common or external iliac arteries (57) (Fig 15).

Another important consideration is the distance between the edge of the tumor and the celiac artery at the level of the origin of the splenic artery for cancers arising in the pancreatic body and/or tail. In a retrospective analysis of 52 consecutive cases of distal pancreatectomy for PDAC, investigators showed a higher incidence of positive resection margin (R1) in tumors located 10 mm or less from the origin of the splenic artery from the celiac artery, compared with patients who underwent distal pancreatectomy with celiac artery resection (53). Therefore, measurement of this distance is important for determining the prognosis of these patients.

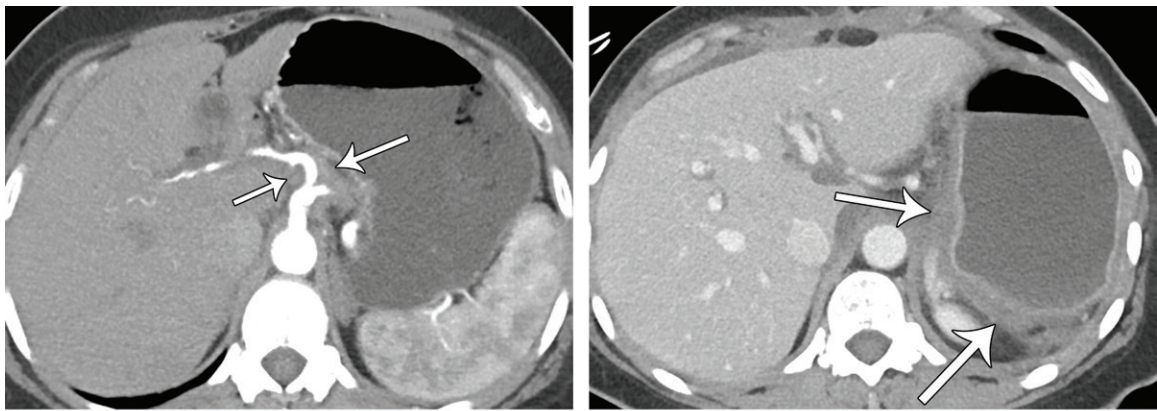
Celiac artery stenosis is observed in 2.0%–7.6% of patients undergoing pancreaticoduodenectomy (58) and may affect the surgical approach in these patients. Ninety percent of cases of celiac artery stenosis are caused by external compression by the median arcuate ligament, a condition referred to as median arcuate ligament syndrome, followed



a.

b.

Figure 14. PDAC involving the pancreatic body and tail in a 64-year-old man. (a) Preoperative axial contrast-enhanced CT image shows encasement of the celiac axis (thin arrows) by the pancreatic mass, along with distal pancreatic atrophy (thick arrow). (b) Axial contrast-enhanced CT image obtained 2 months after a modified Appleby procedure with resection of the pancreatic tail, splenectomy, and arterial reconstruction of the celiac axis and the common hepatic artery with a cadaveric jump graft shows a patent hepatic artery jump graft (arrow).



a.

b.

Figure 15. PDAC involving the pancreatic body and tail in a 40-year-old woman. (a) Axial contrast-enhanced CT image obtained in the arterial phase shows tumor encasement (arrows) of the celiac axis and the hepatic artery. The patient underwent distal pancreatectomy and splenectomy with celiac artery resection and reconstruction with a bifurcated 6-mm ringed jump graft. (b) Postoperative volume-rendered CT image shows the bifurcated jump graft between the right common iliac artery (thick arrow) and the right and left hepatic arteries (thin arrows). (c) Axial contrast-enhanced CT image shows gastric distention with a thickened wall (arrows), findings indicative of ischemic gastritis, at 1 month after surgery. The findings were due to the compromised blood supply caused by the loss of the left gastric artery.



b.

by internal occlusion with atherosclerotic plaque (59). In the presence of celiac axis involvement when pancreaticoduodenectomy is required, median arcuate ligament release is important because the gastroduodenal artery is resected during this operation, with reliance on flow to the liver via the celiac axis. For distal pancreatectomy, the gastroduodenal artery is left intact, and usually celiac artery stenosis is overcome by the development of a collateral pathway between the common hepatic artery and the SMA through retrograde flow via the gastroduodenal artery and the arc of Buhler (60). In 2012, Sugae et al (61) proposed a mor-

phologic grading system to describe the degree of median arcuate ligament compression, a system that could help in predicting the procedure that

Table 3: Classification of Celiac Axis Stenosis Caused by Median Arcuate Ligament Compression, as Proposed by Sugae et al (61)

Classification Criteria	Type A	Type B	Type C
Rate of stenosis (Fig 16, line A/line B) (%)	<50	50–80	80–100
Length of stenosis (Fig 16, line C) (mm)	<3	3–8	>8
Distance from the aorta (Fig 16, line D) (mm)	≥5	NA	<5
Collateral pathways	No	Small periampullary	Large arcade between IPDA and GDA
Expected procedure	None	Median arcuate ligament division	Arterial reconstruction to preserve the collateral pathways

Note.—GDA = gastroduodenal artery, IPDA = inferior pancreaticoduodenal artery, NA = not applicable.

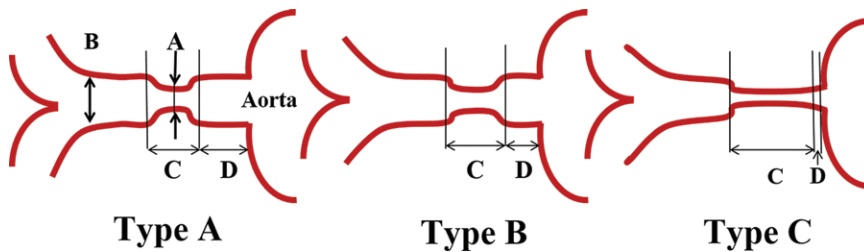


Figure 16. Diagrams of the classification of celiac axis stenosis caused by median arcuate ligament compression into three types, as proposed by Sugae et al (61): type A, diameter of the stenosis (A); type B, diameter of the normal portion of the vessel (B, double-headed arrow on type A diagram); and type C, length of the stenosis (C). D = distance of stenosis from the aorta.

may be required during surgery to release the compression on the celiac artery; the system is based on the stenosis rate and length, the distance of the stenotic segment from the aorta, and the presence or absence of a collateral pathway (Table 3, Fig 16). In addition, the presence of hepatic artery pulsation is first assessed during surgery after clamping of the gastroduodenal artery, with celiac artery revascularization being considered if the pulsation is diminished even after release of the median arcuate ligament compression (Fig 17). Therefore, in addition to a description of the tumor-vessel interface (abutment or encasement), a comprehensive description of the relationship between the tumor and the celiac artery, including the patency of the gastroduodenal artery, the distance between the tumor edge and the origin of the celiac artery from the aorta (which serves as the proximal stump that is clamped and either ligated or reconstructed), and the distance between the tumor edge and the celiac artery at the level of the origin of the splenic artery are part of the preoperative evaluation for PDAC resection.

SMA Involvement

PDAC frequently invades the lymphatic vessels and the periarterial neural plexus once it extends to the extrapancreatic fatty tissue. Tumor adherent to the SMA may invade its rich periarterial lymphatic and neural plexus and may increase the

risk of recurrent disease (62). Radical resection frequently yields positive margins (4) and remains controversial (5). In addition to the SMA being a major blood supplier to the bowel, resection of the sympathetic nerve plexus of the intestinal tract around the SMA may result in intractable diarrhea because of rapid gastrointestinal transit. Consensus exists on abutment of the tumor-SMA interface as a cutoff that distinguishes borderline resectable from locally advanced unresectable disease (26,52,63). Variations in the anatomic configuration of the SMA, particularly those related to the origin of the hepatic artery, are important considerations for preoperative planning.

Anatomic Variants of the Peripancreatic SMA and Hepatic Artery That May Interfere with Resection

Pancreaticoduodenectomy is a technically challenging surgical procedure associated with high morbidity and mortality (64). Furthermore, pancreaticoduodenectomy in the presence of anatomic arterial variants increases the risk of vascular complications, and preoperative imaging may help identify such variants. Thus, evaluation of anatomic arterial variants is an important part of the preoperative evaluation before pancreaticoduodenectomy because preventing an intraoperative vascular injury reduces the risk of postoperative complications such as ischemia, hemorrhage, biliary anastomotic

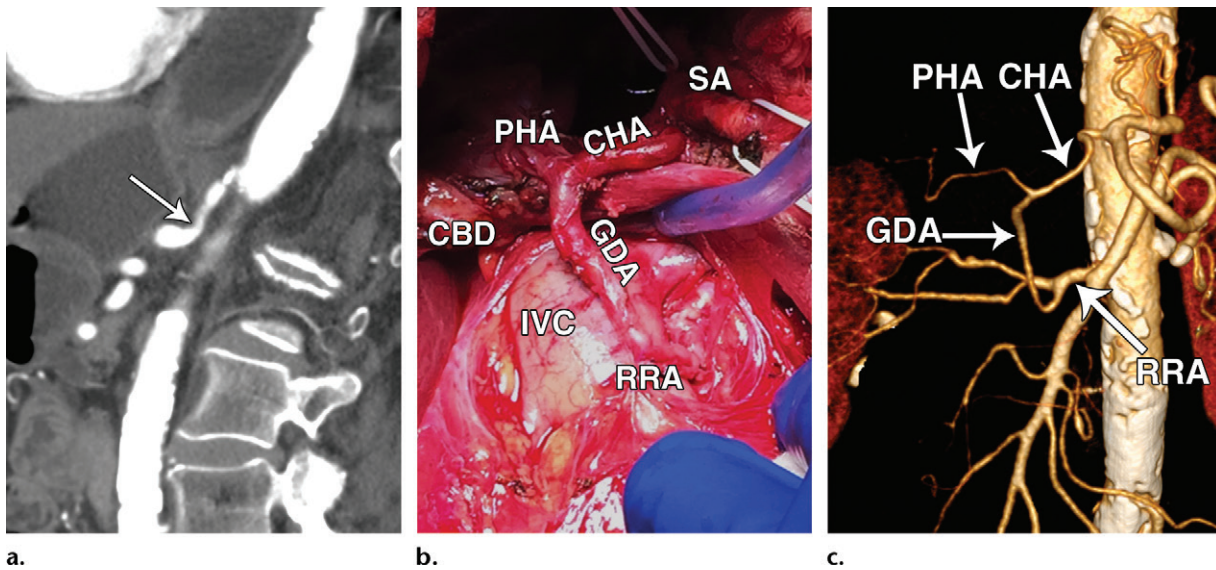


Figure 17. PDAC in the pancreatic head in a 78-year-old woman. (a) Sagittal CT image shows celiac artery stenosis (arrow) with poststenotic dilatation. During surgery, the common hepatic artery was hypotrophic, the gastroduodenal artery was engorged, and when clamped, the proper hepatic artery pulse notably diminished, findings that are indicative of dependence on blood flow to the liver through the gastroduodenal artery. (b) Intraoperative photograph after pancreaticoduodenectomy was performed shows an end-to-side anastomosis between the gastroduodenal artery (GDA) and the right renal artery (RRA). CBD = common bile duct, CHA = common hepatic artery, IVC = inferior vena cava, PHA = proper hepatic artery, SA = splenic artery. (c) Postoperative volume-rendered CT image shows the successful arterial reconstruction 1 month after the Whipple procedure. CHA = common hepatic artery, GDA = gastroduodenal artery, PHA = proper hepatic artery, RRA = right renal artery.

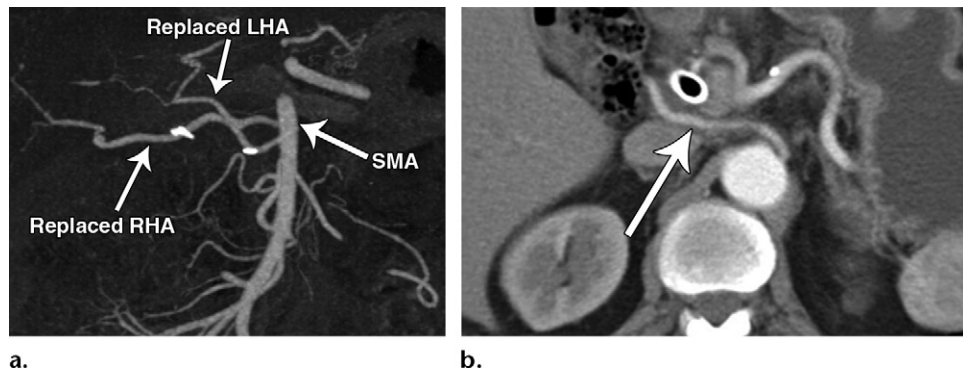


Figure 18. Peripancreatic arterial anatomic variants in two patients. A replaced right hepatic artery is one of the most common anatomic variants in the peripancreatic arterial structures. (a) Maximum intensity projection CT image of a 67-year-old man shows a replaced right hepatic artery (RHA) and a replaced left hepatic artery (LHA) originating from the SMA. (b) Axial CT image of a 66-year-old woman shows a replaced right hepatic artery (arrow) arising from the aorta.

leak, or pseudoaneurysm formation (58,65). The classic anatomic structures of the peripancreatic arteries and their variants have been described in the literature (66). An anatomic variant of the hepatic arterial system is seen in 55%–79% of the patients (58). The most common anatomic variants of the hepatic arterial system are a replaced right hepatic artery, a replaced left hepatic artery, and an accessory right hepatic artery or left hepatic artery. Although the aberrant vessels should be preserved during resection, variants such as accessory hepatic arteries and the replaced left hepatic artery arising from the left gastric artery usually do not determine resectability (67).

Replaced Right Hepatic Artery.—A replaced right hepatic artery is the most common hepatic arterial anatomic variant. The rate of occurrence of this variant, in which the proper hepatic artery gives off only the left hepatic artery while the right hepatic artery originates from the SMA to pass posterolateral to the portal vein, has been reported in the literature to range from 11% to 21% (68) (Fig 18). A replaced right hepatic artery has also been reported to travel behind or through the pancreatic head, predisposing it to tumor extension and/or iatrogenic injury. Because the right hepatic artery is the major source of blood supply to the bile ducts, injury to the replaced right hepatic

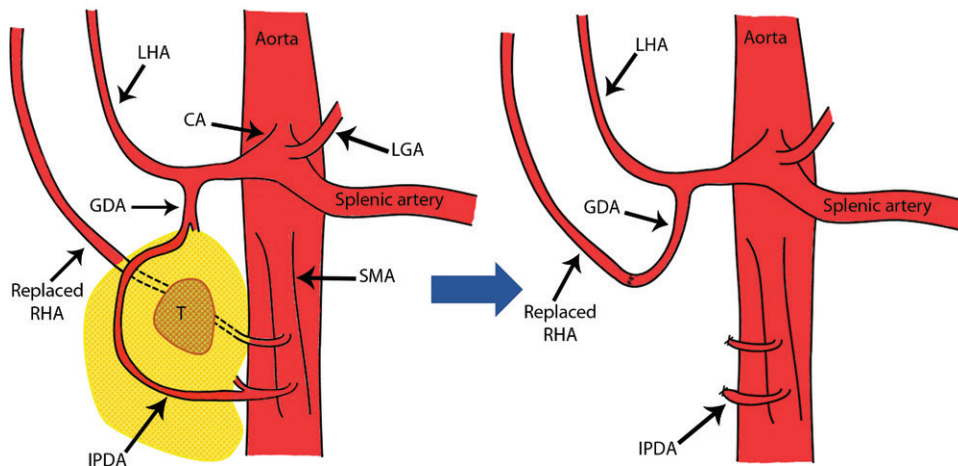


Figure 19. Diagrams show reconstruction of a replaced right hepatic artery by using the gastroduodenal artery transposition technique. The tumor (*T*) in the pancreatic head involves the replaced right hepatic artery (*RHA*). The distal stump of the replaced right hepatic artery is anastomosed with the proximal stump of the gastroduodenal artery (*GDA*) to reconstitute the right hepatic artery blood supply. *CA* = celiac artery, *IPDA* = inferior pancreaticoduodenal artery, *LGA* = left gastric artery, *LHA* = left hepatic artery.

artery may affect the bile duct vascularity, with a subsequent leak from the bilioenteric anastomosis (69). Several methods have been described for reconstruction of the replaced right hepatic artery in case of resection, including venous and prosthetic graft interposition, as well as a gastroduodenal artery transposition technique in which the distal stump of the replaced right hepatic artery is anastomosed with the proximal stump of the gastroduodenal artery (70) (Fig 19).

A potential pitfall in the identification of a replaced right hepatic artery is in the presence of celiac artery stenosis. In such cases, the inferior pancreaticoduodenal artery may become enlarged and may be confused with a replaced right hepatic artery. In other cases, there may be a common origin of the inferior pancreaticoduodenal artery and the replaced right hepatic artery from the SMA (71). Therefore, it is important to follow the first branch arising from the right side of the SMA and see whether it ascends to the porta hepatis (replaced right hepatic artery) or communicates with the superior pancreaticoduodenal artery from the gastroduodenal artery (inferior pancreaticoduodenal artery).

Replaced Common Hepatic Artery.—Instead of arising from the celiac axis, the common hepatic artery may originate from the SMA by a common trunk referred to as the hepatomesenteric trunk, a variant with an incidence ranging from 0.4% to 4.5% (58). The replaced common hepatic artery runs through the pancreatic parenchyma or ascends behind the head of the pancreas and then reaches the porta hepatis medial to the common bile duct in the usual location of the gastroduodenal artery (65). Accidental ligation can lead to a biliary leak

caused by ischemia to the bilioenteric anastomosis, as well as liver ischemia and necrosis (69). The common hepatic artery may less commonly originate from the left gastric artery (68), but this configuration would not interfere with the surgical approach except in cases of a large pancreatic neck mass, which requires celiac axis resection.

Common Trunks.—A common hepatic trunk arising from the SMA, which is termed a hepatomesenteric trunk, may course through the pancreatic parenchyma, and its ligation can lead to hepatic necrosis (72). A celioesenteric trunk is a rare variant with the celiac artery and the SMA arising from the aorta as a common trunk (73). Multidetector CT angiography is superior to conventional angiography because of the added information it provides about the course of the vessel within the pancreatic parenchyma.

Radiographic Evaluation of the Neoadjuvant Response

Preoperative Therapy

Patients with borderline resectable PDAC are treated with preoperative or neoadjuvant therapy to increase the potential of decreasing the stage of the tumor (tumor downstaging) and to achieve an R0 resection (tumor negative) from R1 (microscopic residual tumor) or R2 (macroscopic residual tumor) disease. Treatment typically consists of a combination of chemotherapy and radiation therapy, and the treatment regimens are usually based on the experience of individual institutions. Combined treatment with chemotherapy and radiation therapy allows tumor downstaging in about 30% of patients (7).



Figure 20. PDAC in the pancreatic head in a 54-year-old man. *PV* = portal vein. (a) Coronal contrast-enhanced CT image shows a pancreatic head tumor (*T*) interfacing with the portal vein, without luminal narrowing. (b) Coronal contrast-enhanced CT image shows the tumor (*T*) in the pancreatic head growing in size after 4 months of neoadjuvant chemotherapy. Margin-positive resection of the mass and total pancreatectomy were performed. (c) Coronal contrast-enhanced CT image obtained 4 months after surgery shows a recurrent mass (*R*) in the surgical bed, with narrowing of the SMV (arrow).

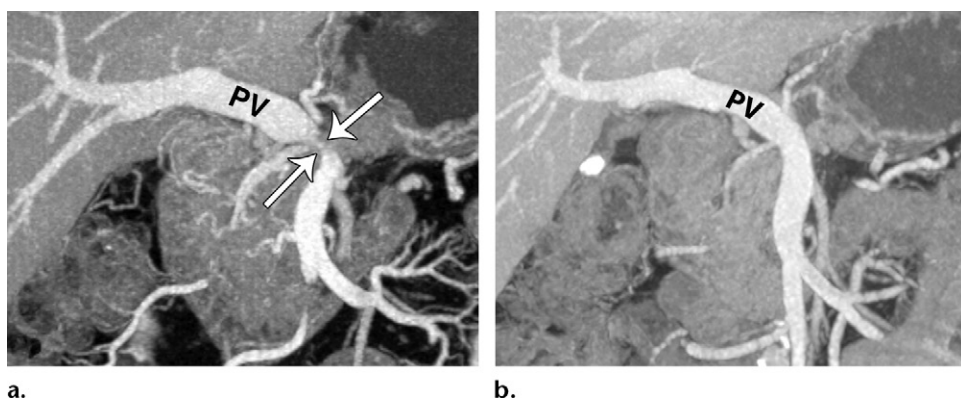


Figure 21. PDAC in the pancreatic head in a 71-year-old man. *PV* = portal vein. (a) Coronal contrast-enhanced CT maximum intensity projection image shows a mass in the pancreatic head, with nearly complete occlusion (arrows) of the portal vein. The patient received neoadjuvant chemoradiation therapy for 5 months. (b) Coronal contrast-enhanced maximum intensity projection CT image obtained after the patient completed the preoperative treatment shows that the portal vein has regained its normal caliber. The patient underwent successful resection, with negative resection margins.

Patients who develop progressive disease during preoperative therapy may be spared unnecessary surgery because they likely have aggressive tumors with poor outcomes (Fig 20).

Multidetector CT is used to evaluate tumor behavior according to the change in tumor size, the tumor-vessel relationship, and the development of distant metastasis during preoperative neoadjuvant treatment of PDAC (Fig 21). A problem with using tumor size as a marker for treatment response is the development of fibrosis and scar tissue after the administration of neoadjuvant therapy, findings that are difficult to distinguish from the residual tumor by using multidetector CT alone. In a study by Katz et al (74), no association was seen between the change in tumor size at multidetector CT according to the Response Evaluation Criteria in Solid Tumors (RECIST) and postoperative outcomes. A similar problem exists when comparing attenuation differences before and after neoadjuvant therapy. In a recent prospec-

tive study that included 47 patients with locally advanced pancreatic adenocarcinoma who received neoadjuvant chemoradiation therapy and underwent surgical exploration, the multidetector CT images obtained before and after neoadjuvant therapy were reviewed, and a score was given for tumor contact with the portal vein and/or SMV, with the hepatic artery, and with the celiac artery (0, no contact; 1, <50% contact; 2, \geq 50% contact) and for the tumor-SMA contact (0, no contact; 1, <25% contact; 2, 25% to <50% contact; 3, \geq 50% contact) (75). Narrowing of the portal vein and/or SMV was also graded (0, absent; grade 1, stenosis of <50%; grade 2, stenosis of \geq 50%). One point or more decrease in the tumor-vessel circumferential contact was found to be associated with higher rates of a negative resection margin (R0), with a specificity of 86%, sensitivity of 61%, positive predictive value of 91%, and negative predictive value of 48%. The change in the tumor size was not significantly associated with R0 resection

(75). Multidetector CT evaluation may suffer from decreased specificity in determining the operability and R0 resectability of PDAC after preoperative treatment because of overestimation of the tumor size and vascular involvement (76).

Diffusion-weighted MR imaging has shown promise in predicting the response to chemoradiation therapy. Tumors with a low value for the apparent diffusion coefficient at baseline may respond poorly to chemoradiation therapy because of the presence of dense fibrosis that limits the delivery of radiosensitizing systemic therapy and requires more aggressive treatment (77). In a study of the neoadjuvant treatment response evaluated by assessing the change in the maximum standard uptake value (SUV_{max}) for locally advanced PDAC by using ^{18}F -fluorodeoxyglucose PET with CT and providing anatomic data (PET and CT) according to the European Organisation for Research and Treatment of Cancer (EORTC) guidelines (78), investigators showed that the pre- and posttreatment SUV_{max} difference was an independent predictor of the postoperative clinical outcomes, including longer median overall survival, progression-free survival, and local-regional progression-free survival (79). Two nonradiologic factors routinely used to evaluate tumor response to neoadjuvant therapy are a change in the serum carbohydrate antigen 19-9 (80) and improved performance status (52).

Imaging Report

Imaging plays a vital role in the initial staging and the decision-making process for patients with PDAC. Considerable variability exists in the terms used by radiologists to describe the extent of disease. Furthermore, freestyle narrative reports can potentially be confusing because the critical information may not be presented in a concise manner that is understandable to all members of the multidisciplinary clinical team. Although the surgeons need to assess the extent of the disease at preoperative imaging themselves and decide on the surgical approach on the basis of the imaging findings, it is nevertheless vital to have a comprehensive and uniform reporting system by which key descriptors that define disease extent can be conveyed not only to the surgeons but also to other members of the team, including the oncologists, radiation oncologists, and gastroenterologists, for appropriate management of the disease.

Apart from the clinically relevant features of PDAC described herein, interest has been growing in the use of standardized reporting templates for clinical and research purposes. Although such initiatives have been successful

in other domains, such as for prostate cancer, breast imaging, and musculoskeletal imaging, structured reporting for PDAC remains contentious because of concerns about a lack of completeness, the rigidity of the report templates, and the increased time consumption (81,82). The superiority of a structured radiologic template, compared with the free text style, in reporting the multidetector CT findings has been previously addressed (83). Brook et al (84) compared structured reporting to descriptive reporting and concluded that the use of structured reports for evaluation of PDAC provides superior evaluation of PDAC for surgical planning by improving confidence among surgeons for decisions about treatment paradigms. In this study by Brook et al (84), structured reporting provided an increased number of key descriptors, such as tumor staging, vascular thrombosis, aberrant vascular anatomic structures, and the presence of atherosclerotic disease of the celiac axis. Also, the information was more easily extracted by the referring physician.

A standardized imaging reporting template was proposed by a multi-institutional group of PDAC experts under the joint sponsorship of the Society of Abdominal Radiology and the American Pancreatic Association (50). The template consists of morphologic evaluation of the pancreatic tumor, as well as vascular (both arterial and venous) and extrapancreatic evaluation, as summarized in Table 4 (50). The proposed PDAC staging template is comprehensive because it includes not only the findings described in the National Comprehensive Cancer Network guidelines for criteria for resectability (63) but also other findings that may change the surgical plan of resection and/or reconstruction (eg, extension of the tumor to the origin of the right hepatic artery and left hepatic artery, extension to the veins draining into the SMV, and the presence of arterial variants). Template-based reporting should help decrease the incomplete documentation of descriptors that define the tumor-vessel relationship, and such incomplete documentation interferes with the decision-making process for patients with PDAC. Adoption of uniform terminology should help with the management decisions for patients with PDAC and promote standardization of research and clinical protocols across institutions.

Conclusion

An understanding of the key descriptors of PDAC and their importance, especially in relation to the perivascular spread of disease, provides essential information that helps determine treatment options. Borderline resectable PDAC is an important clinical entity because of

Table 4: PDAC Radiology Reporting Template from the Consensus Statement of the Society of Abdominal Radiology and the American Pancreatic Association

Category and Parameters	Finding
Morphologic evaluation	
Appearance in the pancreatic parenchymal phase	Hypo-, iso-, or hyperattenuating
Size (axial dimension) (cm)	Measurable or nonmeasurable
Location	Head, uncinate process, body, or tail
Pancreatic duct abrupt narrowing and upstream dilatation	Yes or no
Biliary tree abrupt narrowing and upstream dilatation	Yes or no
Arterial disease (SMA, celiac axis, or common hepatic artery)	
Soft-tissue contact	$\leq 180^\circ$ or $>180^\circ$
Hazy stranding contact	$\leq 180^\circ$ or $>180^\circ$
Vascular contour irregularity	Yes or no
Extension to first SMA branch, hepatic artery bifurcation	Yes or no
Anatomic variants	Yes or no; describe
Venous disease (portal vein and/or SMV)	
Soft-tissue contact	$\leq 180^\circ$ or $>180^\circ$
Hazy stranding contact	$\leq 180^\circ$ or $>180^\circ$
Vascular contour irregularity (tethering or teardrop)	Yes or no
Extension to first draining vein	Yes or no
Venous thrombosis	Yes or no; bland or tumor thrombus
Venous collateral vessels	Yes or no
Extrapancreatic disease	
Liver lesions	Yes or no; suspicious, indeterminate, or benign
Peritoneal or omental disease	Yes or no
Ascites	Yes or no
Suspicious lymph nodes	Yes or no
Other disease	Yes or no; describe

Source.—Reference 50.

the large percentage of patients presenting with stage III disease and the increasing number of treatment options available for such patients. With the evolution of newer surgical and chemoradiation therapy options, the definition of borderline resectable disease continues to evolve; and optimized accuracy of preoperative staging for metastases and an accurate assessment of disease response, especially to new promising chemotherapeutic agents, are high-yield areas for affecting patient outcomes. A comprehensive description of the relationship between PDAC and the peripancreatic vessels and their variants can be obtained by careful evaluation of the multidetector CT images in multiple planes to include all of the descriptors that surgeons may need for surgical planning and for the administration of neoadjuvant therapy. The use of standardized terminology and a radiologic template to document the multidetector CT findings for PDAC can limit the subjective evaluation of the tumor-vessel relationship and mandates the provision of key information that may otherwise be missed.

References

1. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2016. *CA Cancer J Clin* 2016;66(1):7–30.
2. Seufferlein T, Bachet JB, Van Cutsem E, Rougier P; ESMO Guidelines Working Group. Pancreatic adenocarcinoma: ESMO-ESDO clinical practice guidelines for diagnosis, treatment and follow-up. *Ann Oncol* 2012;23(suppl 7):vii33–vii40.
3. Allema JH, Reinders ME, van Gulik TM, et al. Portal vein resection in patients undergoing pancreatoduodenectomy for carcinoma of the pancreatic head. *Br J Surg* 1994;81(11):1642–1646.
4. Fuhrman GM, Leach SD, Staley CA, et al; Pancreatic Tumor Study Group. Rationale for *en bloc* vein resection in the treatment of pancreatic adenocarcinoma adherent to the superior mesenteric–portal vein confluence. *Ann Surg* 1996;223(2):154–162.
5. Yekebas EF, Bogoevski D, Cataldegirmen G, et al. *En bloc* vascular resection for locally advanced pancreatic malignancies infiltrating major blood vessels: perioperative outcome and long-term survival in 136 patients. *Ann Surg* 2008;247(2):300–309.
6. Neoptolemos JP, Stocken DD, Friess H, et al. A randomized trial of chemoradiotherapy and chemotherapy after resection of pancreatic cancer. *N Engl J Med* 2004;350(12):1200–1210.
7. Gillen S, Schuster T, Meyer Zum Büschenfelde C, Friess H, Kleeff J. Preoperative/neoadjuvant therapy in pancreatic cancer: a systematic review and meta-analysis of response and resection percentages. *PLoS Med* 2010;7(4):e1000267. doi:10.1371/journal.pmed.1000267. Published online April 20, 2010.

8. Chu CK, Farnell MB, Nguyen JH, et al. Prosthetic graft reconstruction after portal vein resection in pancreaticoduodenectomy: a multicenter analysis. *J Am Coll Surg* 2010;211(3):316–324.
9. Zureikat AH, Nguyen KT, Bartlett DL, Zeh HJ, Moser AJ. Robotic-assisted major pancreatic resection and reconstruction. *Arch Surg* 2011;146(3):256–261.
10. Cauley CE, Waters JA, Dumas RP, et al. Outcomes of primary surveillance for intraductal papillary mucinous neoplasm. *J Gastrointest Surg* 2012;16(2):258–267; discussion 266.
11. Valls C, Andia E, Sanchez A, et al. Dual-phase helical CT of pancreatic adenocarcinoma: assessment of resectability before surgery. *AJR Am J Roentgenol* 2002;178(4):821–826.
12. Tunaci M. Multidetector row CT of the pancreas. *Eur J Radiol* 2004;52(1):18–30.
13. Ishikawa O, Ohigashi H, Imaoka S, et al. Preoperative indications for extended pancreatotomy for locally advanced pancreas cancer involving the portal vein. *Ann Surg* 1992;215(3):231–236.
14. Tran Cao HS, Balachandran A, Wang H, et al. Radiographic tumor-vein interface as a predictor of intraoperative, pathologic, and oncologic outcomes in resectable and borderline resectable pancreatic cancer. *J Gastrointest Surg* 2014;18(2):269–278; discussion 278.
15. Fletcher JG, Wiersma MJ, Farrell MA, et al. Pancreatic malignancy: value of arterial, pancreatic, and hepatic phase imaging with multi-detector row CT. *Radiology* 2003;229(1):81–90.
16. Horton KM, Fishman EK. Adenocarcinoma of the pancreas: CT imaging. *Radiol Clin North Am* 2002;40(6):1263–1272.
17. Treadwell JR, Zafar HM, Mitchell MD, Tipton K, Teitelbaum U, Jue J. Imaging tests for the diagnosis and staging of pancreatic adenocarcinoma: a meta-analysis. *Pancreas* 2016;45(6):789–795.
18. Chen FM, Ni JM, Zhang ZY, Zhang L, Li B, Jiang CJ. Presurgical evaluation of pancreatic cancer: a comprehensive imaging comparison of CT versus MRI. *AJR Am J Roentgenol* 2016;206(3):526–535.
19. Park HS, Lee JM, Choi HK, Hong SH, Han JK, Choi BI. Preoperative evaluation of pancreatic cancer: comparison of gadolinium-enhanced dynamic MRI with MR cholangiopancreatography versus MDCT. *J Magn Reson Imaging* 2009;30(3):586–595.
20. Koelblinger C, Ba-Ssalamah A, Goetzinger P, et al. Gadobenate dimeglumine-enhanced 3.0-T MR imaging versus multiphase 64-detector row CT: prospective evaluation in patients suspected of having pancreatic cancer. *Radiology* 2011;259(3):757–766.
21. Tempero MA, Arnoletti JP, Behrman SW, et al. Pancreatic adenocarcinoma, version 2.2012: featured updates to the NCCN guidelines. *J Natl Compr Canc Netw* 2012;10(6):703–713.
22. Choi HJ, Kang CM, Lee WJ, et al. Prognostic value of ¹⁸F-fluorodeoxyglucose positron emission tomography in patients with resectable pancreatic cancer. *Yonsei Med J* 2013;54(6):1377–1383.
23. Im HJ, Oo S, Jung W, et al. Prognostic value of metabolic and volumetric parameters of preoperative FDG-PET/CT in patients with resectable pancreatic cancer. *Medicine (Baltimore)* 2016;95(19):e3686. doi:10.1097/MD.0000000000003686. Published online May 13, 2016.
24. McIntyre CA, Winter JM. Diagnostic evaluation and staging of pancreatic ductal adenocarcinoma. *Semin Oncol* 2015;42(1):19–27.
25. Vauthey JN, Dixon E. AHPBA/SSO/SSAT Consensus Conference on Resectable and Borderline Resectable Pancreatic Cancer: rationale and overview of the conference. *Ann Surg Oncol* 2009;16(7):1725–1726.
26. Katz MH, Marsh R, Herman JM, et al. Borderline resectable pancreatic cancer: need for standardization and methods for optimal clinical trial design. *Ann Surg Oncol* 2013;20(8):2787–2795.
27. Nakao A, Kanzaki A, Fujii T, et al. Correlation between radiographic classification and pathological grade of portal vein wall invasion in pancreatic head cancer. *Ann Surg* 2012;255(1):103–108.
28. Tseng JF, Raut CP, Lee JE, et al. Pancreaticoduodenectomy with vascular resection: margin status and survival duration. *J Gastrointest Surg* 2004;8(8):935–949; discussion 949–950.
29. Giovino F, Turri G, Katz MH, Heaton N, Ahmed I. Meta-analysis of benefits of portal-superior mesenteric vein resection in pancreatic resection for ductal adenocarcinoma. *Br J Surg* 2016;103(3):179–191.
30. Callery MP, Chang KJ, Fishman EK, Talamonti MS, Traverso LW, Linehan DC. Pretreatment assessment of resectable and borderline resectable pancreatic cancer: expert consensus statement. *Ann Surg Oncol* 2009;16(7):1727–1733.
31. Akasbi Y, Arifi S, El Idrissi M, et al. Soft-tissue metastasis revealing a pancreatic adenocarcinoma: one case report and a review of literature. *Pan Afr Med J* 2012;11:32. doi:10.11604/pamj.2012.11.32.776. Published online February 22, 2012.
32. 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):1439–1443.
33. Loyer EM, David CL, Dubrow RA, Evans DB, Charnsangavej C. Vascular involvement in pancreatic adenocarcinoma: reassessment by thin-section CT. *Abdom Imaging* 1996;21(3):202–206.
34. Vargas R, Nino-Murcia M, Trueblood W, Jeffrey RB Jr. MDCT in pancreatic adenocarcinoma: prediction of vascular invasion and resectability using a multiphase technique with curved planar reformations. *AJR Am J Roentgenol* 2004;182(2):419–425.
35. Hough TJ, Raptopoulos V, Siewert B, Matthews JB. Tear-drop superior mesenteric vein: CT sign for unresectable carcinoma of the pancreas. *AJR Am J Roentgenol* 1999;173(6):1509–1512.
36. Springett GM, Hoffe SE. Borderline resectable pancreatic cancer: on the edge of survival. *Cancer Contr* 2008;15(4):295–307.
37. Zakharova OP, Karmazanovsky GG, Egorov VI. Pancreatic adenocarcinoma: outstanding problems. *World J Gastrointest Surg* 2012;4(5):104–113.
38. Kaneoka Y, Yamaguchi A, Isogai M. Portal or superior mesenteric vein resection for pancreatic head adenocarcinoma: prognostic value of the length of venous resection. *Surgery* 2009;145(4):417–425.
39. Artifon EL, Chu A, Freeman M, Sakai P, Usmani A, Kumar A. A comparison of the consensus and clinical definitions of pancreatitis with a proposal to redefine post-endoscopic retrograde cholangiopancreatography pancreatitis. *Pancreas* 2010;39(4):530–535.
40. Kaneoka Y, Maeda A, Isogai M. Surgical outcome of autologous external iliac vein grafting in cases of hepatopancreato-biliary malignancy: how I do it. *J Gastrointest Surg* 2012;16(8):1590–1596.
41. Miyazaki M, Itoh H, Kaiho T, et al. Portal vein reconstruction at the hepatic hilus using a left renal vein graft. *J Am Coll Surg* 1995;180(4):497–498.
42. Clagett GP, Bowers BL, Lopez-Viego MA, et al. Creation of a neo-aortoiliac system from lower extremity deep and superficial veins. *Ann Surg* 1993;218(3):239–248; discussion 248–249.
43. Katz MH, Fleming JB, Pisters PW, Lee JE, Evans DB. Anatomy of the superior mesenteric vein with special reference to the surgical management of first-order branch involvement at pancreaticoduodenectomy. *Ann Surg* 2008;248(6):1098–1102.
44. Yoshimi F, Asato Y, Tanaka R, et al. Reconstruction of the portal vein and the splenic vein in pancreaticoduodenectomy for pancreatic cancer. *Hepatogastroenterology* 2003;50(51):856–860.
45. Pilgrim CH, Tsai S, Tolat P, et al. Optimal management of the splenic vein at the time of venous resection for pancreatic cancer: importance of the inferior mesenteric vein. *J Gastrointest Surg* 2014;18(5):917–921.
46. Clark LR, Jaffe MH, Choyke PL, Grant EG, Zeman RK. Pancreatic imaging. *Radiol Clin North Am* 1985;23(3):489–501.
47. Bachellier P, Rosso E, Lucescu I, et al. Is the need for an arterial resection a contraindication to pancreatic resection for locally advanced pancreatic adenocarcinoma? a case-matched controlled study. *J Surg Oncol* 2011;103(1):75–84.
48. Mollberg N, Rahbari NN, Koch M, et al. Arterial resection during pancreatotomy for pancreatic cancer: a systematic review and meta-analysis. *Ann Surg* 2011;254(6):882–893.
49. Horton KM, Fishman EK. Multidetector CT angiography of pancreatic carcinoma. I. Evaluation of arterial involvement. *AJR Am J Roentgenol* 2002;178(4):827–831.

50. Al-Hawary MM, Francis IR, Chari ST, et al. Pancreatic ductal adenocarcinoma radiology reporting template: consensus statement of the Society of Abdominal Radiology and the American Pancreatic Association. *Radiology* 2014;270(1):248–260.
51. Varadhachary GR, Tamm EP, Abbruzzese JL, et al. Borderline resectable pancreatic cancer: definitions, management, and role of preoperative therapy. *Ann Surg Oncol* 2006;13(8):1035–1046.
52. 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):3409–3413.
53. Okada K, Kawai M, Tani M, et al. Surgical strategy for patients with pancreatic body/tail carcinoma: who should undergo distal pancreatectomy with en-bloc celiac axis resection? *Surgery* 2013;153(3):365–372.
54. Makary MA, Fishman EK, Cameron JL. Resection of the celiac axis for invasive pancreatic cancer. *J Gastrointest Surg* 2005;9(4):503–507.
55. Hirano S, Kondo S, Hara T, et al. Distal pancreatectomy with en bloc celiac axis resection for locally advanced pancreatic body cancer: long-term results. *Ann Surg* 2007;246(1):46–51.
56. Amano H, Miura F, Toyota N, et al. Pancreatectomy with reconstruction of the right and left hepatic arteries for locally advanced pancreatic cancer. *J Hepatobiliary Pancreat Surg* 2009;16(6):777–780.
57. Machado MA, Surjan RC, Nishinari K, Makdissi FF, Machado MC. Iliac-hepatic arterial bypass for compromised collateral flow during modified Appleby operation for advanced pancreatic cancer. *Eur J Surg Oncol* 2009;35(10):1124–1127.
58. Shukla PJ, Barreto SG, Kulkarni A, Nagarajan G, Fingerhut A. Vascular anomalies encountered during pancreatoduodenectomy: do they influence outcomes? *Ann Surg Oncol* 2010;17(1):186–193.
59. Gaujoux S, Sauvanet A, Vullierme MP, et al. Ischemic complications after pancreatoduodenectomy: incidence, prevention, and management. *Ann Surg* 2009;249(1):111–117.
60. Song SY, Chung JW, Kwon JW, et al. Collateral pathways in patients with celiac axis stenosis: angiographic–spiral CT correlation. *RadioGraphics* 2002;22(4):881–893.
61. Sugae T, Fujii T, Kodera Y, et al. Classification of the celiac axis stenosis owing to median arcuate ligament compression, based on severity of the stenosis with subsequent proposals for management during pancreatoduodenectomy. *Surgery* 2012;151(4):543–549.
62. Nagakawa T, Mori K, Nakano T, et al. Perineural invasion of carcinoma of the pancreas and biliary tract. *Br J Surg* 1993;80(5):619–621.
63. Tempero MA, Malafa MP, Behrman SW, et al. Pancreatic adenocarcinoma, version 2.2014: featured updates to the NCCN guidelines. *J Natl Compr Canc Netw* 2014;12(8):1083–1093.
64. Yeo CJ, Cameron JL, Sohn TA, et al. Six hundred fifty consecutive pancreatoduodenectomies in the 1990s: pathology, complications, and outcomes. *Ann Surg* 1997;226(3):248–257; discussion 257–260.
65. Volpe CM, Peterson S, Hoover EL, Doerr RJ. Justification for visceral angiography prior to pancreatoduodenectomy. *Am Surg* 1998;64(8):758–761.
66. Covey AM, Brody LA, Maluccio MA, Getrajdman GI, Brown KT. Variant hepatic arterial anatomy revisited: digital subtraction angiography performed in 600 patients. *Radiology* 2002;224(2):542–547.
67. Sakorafas GH, Friess H, Balsiger BM, Büchler MW, Sarr MG. Problems of reconstruction during pancreatoduodenectomy. *Dig Surg* 2001;18(5):363–369.
68. Hiatt JR, Gabbay J, Busuttill RW. Surgical anatomy of the hepatic arteries in 1000 cases. *Ann Surg* 1994;220(1):50–52.
69. Traverso LW, Freeny PC. Pancreatoduodenectomy: the importance of preserving hepatic blood flow to prevent biliary fistula. *Am Surg* 1989;55(7):421–426.
70. Allendorf JD, Bellemare S. Reconstruction of the replaced right hepatic artery at the time of pancreatoduodenectomy. *J Gastrointest Surg* 2009;13(3):555–557.
71. Cherian PT, Hegab B, Oliff SP, Wigmore SJ. The management of an accessory or replaced right hepatic artery during multiorgan retrieval: results of an angiographic study. *Liver Transpl* 2010;16(6):742–747.
72. Furukawa H, Shimada K, Iwata R, Moriyama N. A replaced common hepatic artery running through the pancreatic parenchyma. *Surgery* 2000;127(6):711–712.
73. Lawler LP, Fishman EK. Celiomesenteric anomaly demonstration by multidetector CT and volume rendering. *J Comput Assist Tomogr* 2001;25(5):802–804.
74. Katz MH, Fleming JB, Bhosale P, et al. Response of borderline resectable pancreatic cancer to neoadjuvant therapy is not reflected by radiographic indicators. *Cancer* 2012;118(23):5749–5756.
75. Cassinotto C, Mouries A, Lafourcade JP, et al. Locally advanced pancreatic adenocarcinoma: reassessment of response with CT after neoadjuvant chemotherapy and radiation therapy. *Radiology* 2014;273(1):108–116.
76. Cassinotto C, Cortade J, Belleannée G, et al. An evaluation of the accuracy of CT when determining resectability of pancreatic head adenocarcinoma after neoadjuvant treatment. *Eur J Radiol* 2013;82(4):589–593.
77. Cuneo KC, Chenevert TL, Ben-Josef E, et al. A pilot study of diffusion-weighted MRI in patients undergoing neoadjuvant chemoradiation for pancreatic cancer. *Transl Oncol* 2014;7(5):644–649.
78. Siegel R, Naishadham D, Jemal A. Cancer statistics, 2012. *CA Cancer J Clin* 2012;62(1):10–29.
79. Topkan E, Parlak C, Kotek A, Yapar AF, Pehlivan B. Predictive value of metabolic ¹⁸F-FDG-PET response on outcomes in patients with locally advanced pancreatic carcinoma treated with definitive concurrent chemoradiotherapy. *BMC Gastroenterol* 2011;11:123. doi:10.1186/1471-230X-11-123. Published online November 10, 2011.
80. Boone BA, Steve J, Zenati MS, et al. Serum CA 19-9 response to neoadjuvant therapy is associated with outcome in pancreatic adenocarcinoma. *Ann Surg Oncol* 2014;21(13):4351–4358.
81. Johnson AJ, Chen MY, Swan JS, Applegate KE, Littenberg B. Cohort study of structured reporting compared with conventional dictation. *Radiology* 2009;253(1):74–80.
82. Weiss DL, Langlotz CP. Structured reporting: patient care enhancement or productivity nightmare? *Radiology* 2008;249(3):739–747.
83. Schwartz LH, Panicek DM, Berk AR, Li Y, Hricak H. Improving communication of diagnostic radiology findings through structured reporting. *Radiology* 2011;260(1):174–181.
84. Brook OR, Brook A, Vollmer CM, Kent TS, Sanchez N, Pedrosa I. Structured reporting of multiphasic CT for pancreatic cancer: potential effect on staging and surgical planning. *Radiology* 2015;274(2):464–472.