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Differentiating Autoimmune Pancreatitis from Pancreatic Adenocarcinoma using Dual-phase Computed Tomography: An Inter-observer Study

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Abstract

Objective—To study features on dual phase CT that help differentiate autoimmune pancreatitis (AIP) from pancreatic adenocarcinoma (PA).

Methods—The CTs of 32 patients with AIP were matched with equal number of PA and independently evaluated by 3 radiologists who assigned a diagnosis of AIP, PA, or unsure. Interobserver agreement between radiologists was evaluated using kappa statistics.

Results—The mean accuracy for diagnosing AIP and PA was 68% and 83%, respectively. There was moderate agreement between radiologists (κ : 0.58, $p < 0.0001$). The most common findings for AIP: CBD stricture (63%), bile duct wall hyperenhancement (47%), and diffuse parenchymal enlargement (41%). The most common findings for PA: Focal mass (78%, κ : 0.58, $p < 0.0001$) and pancreatic ductal dilatation (69%, κ : 0.7, $p < 0.0001$). Findings helpful for diagnosing AIP were diffuse enlargement, parenchymal atrophy as well as absence of pancreatic duct dilatation and focal mass. Findings helpful for diagnosing PA were focal mass and pancreatic ductal dilatation. Misdiagnosis of PA in patients with AIP was due to focal mass, pancreatic duct dilatation and pancreatic atrophy and AIP in patients with PA was due to absence of atrophy, presence of diffuse enlargement and peripancreatic halo.

Conclusion—Diffuse enlargement, hypoenhancement and characteristic peripancreatic halo are strong indicators for a diagnosis of AIP. Radiologists demonstrated moderate agreement in distinguishing AIP from PA on the basis of CT imaging.

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Keywords

Autoimmune pancreatitis; CT; pancreas adenocarcinoma; differentiation; reader variability

Introduction

Since the proposal of the concept of autoimmune pancreatitis (AIP) by Yoshida et al. in 1995,¹ there has been growing recognition of this disease as a distinct clinical, pathologic, and radiological entity. Unfortunately, AIP can be difficult to distinguish from pancreatic adenocarcinoma (PA) and poses a significant diagnostic dilemma even for experienced radiologists, particularly when lesions are mass forming^{2–4} which may be seen in both type I and type II AIP.⁵ Pancreatic resection may sometimes be performed without histologic proof of malignancy due to the difficulty in imaging differentiation of benign and malignant lesions. Prior studies have shown a prevalence of benign disease in patients undergoing pancreaticoduodenectomy for presumed malignancy to be 8–9%, with nearly one-third attributable to AIP.^{6, 7} As practitioners are becoming more aware of both the clinical and imaging findings, patients undergoing surgery for AIP is decreasing. The imaging differentiation of AIP and pancreatic cancer remains critically important given the vastly different therapeutic and prognostic implications associated with either diagnosis.

Dual phase contrast enhanced computed tomography (CECT) remains a first-line imaging modality for the evaluation of patients with suspected pancreaticobiliary diseases despite recent progress in alternative imaging modalities such as MRI^{8, 9} and PET.¹⁰ AIP is associated with characteristic findings on dual phase CECT that can help distinguish it from PA.^{11–13} However, in clinical practice, due to the overall rarity of both AIP and PA, the presence of mass lesion in the pancreas is usually suspected to be malignant and this might explain why so many patients with AIP undergo surgical resection. Differentiation of AIP from other pancreatic pathology using CT has been limited by small sample size.¹⁴ A recent study evaluated differentiation of mass-forming AIP from PA by combining the findings of CT and MRI but was limited by small sample size and the lack of histologic gold standard for the diagnosis of AIP.¹⁵ Our study describes the imaging findings of a cohort of such patients with a challenging diagnosis and particularly evaluates the findings that not only help in making the correct diagnosis of AIP but also findings that sway the radiologist away from making the right diagnosis. We analyzed the CECT findings of AIP in a group of patients who underwent surgical resection or met the criteria for AIP using the international consensus guidelines,¹⁶ and evaluated the accuracy of a group of experienced blinded radiologists in differentiating AIP from an age and sex matched cohort of patients with histologically confirmed PA.

Methods and Materials

Patient Selection

This retrospective study was approved by our Institutional Review Board with waiver of informed consent and Health Insurance Portability and Accountability Act authorization. Thirty eight patients with AIP were identified through review of an institutional pathology

database using the search terms “autoimmune pancreatitis”, “idiopathic duct centric pancreatitis” and “lymphoplasmacytic sclerosing pancreatitis” between 2001 and 2010. An additional 9 patients evaluated in a specialty pancreatitis clinic in the year 2011 were added for a total of 47 patients with AIP. One patient was under thirty years old and was unable to be matched to a control patient due to outlying age and was thus excluded from the final cohort. Cases referred for pathologic consultation from outside institutions were excluded as diagnostic imaging was not available. Thirty two (M: F 21:11, mean age: 61 years; age range: 36–82 years) patients with a pre-surgical or pre-steroid treatment dual phase CECT were included in the final analysis. The histopathologic diagnosis of AIP was made on the basis of either pancreaticoduodenectomy (n=21), distal pancreatectomy (n=4), Frey procedure (n = 1), or EUS-guided biopsy (n=4). Two patients had equivocal histopathologic findings on endoscopic biopsy but met a sufficient number of other ICDC criteria for the diagnosis of AIP.¹⁶

The control cohort consisted of patients who underwent pancreaticoduodenectomy for PA at our institution between 2001 and 2010 with histologic confirmation of PA. Controls (M: F 21:11, Mean Age: 61; Age Range: 38–82) were age and gender matched to the AIP patients and had a presurgical dual phase CECT. Furthermore, radiology reports were reviewed and patients with a focal mass due to AIP were matched to patients with a mass due to PA in the same location. Patients were also matched on the approximate date of pretreatment imaging to avoid technique and technology differences. There were no PA patients with liver metastases in the study.

Pre-Interventional Imaging

All patients underwent a dual phase CECT within 4 weeks of their surgical resection or biopsy. All CECTs were obtained at our institution and utilized dual phase (arterial and portal venous) intravenous contrast and negative oral contrast (water) as part of a standardized pancreatic or hepatic imaging protocol. Patients were imaged on a range of helical multidetector (4, 16, 64 & 128 slice) CT scanners. All dual phase (arterial and portal venous) pancreatic imaging protocol (kVp 100–120, mAs 100–500) CECTs included an intravenous bolus injection of between 100 and 140 mL of iodinated contrast (Visipaque® 320 or Omnipaque® 350, GE Healthcare, Waukesha WI, U.S.A.) with water as a negative oral contrast. The injection rates were routinely 5 mL/sec with scan delays of 25–30 seconds (arterial phase) and 60 seconds (portal venous phase). Most CTs included both thin (0.75 mm to 1.5 mm) and thick (3 to 5 mm) image series, sagittal and coronal reformatted image series, and 3D post processed images.

Imaging Interpretation

Images were interpreted independently by three radiologists (S.K., S.T., and A.Z., with 14, 3, and 5 years of CT interpretation experience as attending radiologists, respectively) with fellowship training in abdominal imaging. The radiologists were unaware of the histopathology results and were provided with an individual list of accession numbers that had been randomized using the Matlab *rand* function (Mathworks, Natick MA, U.S.A.). Images were retrieved through the Picture Archiving and Communication System (PACS)

using the image accession number to prevent automatic retrieval of prior comparisons. Radiologists were allowed to generate additional multiplanar reformatted series if desired.

A checklist of 14 questions based on findings previously reported in the radiology literature as present in cases of AIP and/or PA was provided to each reviewer (Table 1).^{13, 17–21} Radiologists provided their final diagnosis using a 3 point scale, indicating a diagnosis of AIP, PA or an unsure diagnosis. An option of free-text response for radiologists was provided to indicate most influential findings in the final diagnosis from the provided checklist. A finding was considered to be present when at least two or more radiologists agreed on its presence. Similarly, a finding was considered absent when at least 2 or more radiologists failed to identify it. A consensus on the disagreement on the reading was not made. The relative frequency of specific imaging findings in the AIP and PA groups were determined from these results. In an effort to identify the most influential imaging characteristics discriminating between AIP and PA, the free-text response provided by the radiologists listing the main reason(s) for the particular diagnosis was used. For the purpose of determining whether the influential factors were helpful or misleading, a finding was considered to be helpful when reported as influential by 2 or more radiologists in the presence of a correct diagnosis based on the clinical and pathology results. Similarly, a listed finding was considered to be misleading when an incorrect or unsure final diagnosis was made by 2 or more radiologists.

Statistical Analysis

The frequency of CT findings was calculated on the basis of two or more radiologists regardless of their experience agreeing on a finding. The frequency of findings among AIP and PA patients were compared using a chi square test. A *p*-value of <0.05 was considered statistically significant.

Accuracy for diagnosing AIP or PA was calculated using a crosstab approach for each reader. The reader scoring was evaluated using a 3 point scheme reflecting preference for AIP or PA. Equivocal responses were considered inaccurate for the purposes of calculation. Reader diagnostic responses and responses to the checklist questions were then analyzed with a multi-rater kappa statistics subroutine²² based on the Siegel and Castellan's fixed marginal kappa statistic.²³ The kappa values were interpreted using a scale initially proposed by Landis and Koch.²⁴ Statistical analysis was performed using the SPSS 19 statistical software package (IBM Analytics, Armonk, NY USA).²⁴

RESULTS

Frequency of findings

The most common findings noted among the 32 patients with AIP were common bile duct stricture (63%), hyperenhancement of the bile duct wall (47%), and diffuse parenchymal enlargement (41%) (Figure 1). The most common findings among 32 patients with histologically confirmed PA were focal mass (78%), upstream pancreatic ductal dilatation of >5mm from the mass (69%) and parenchymal atrophy (53%). Diffuse parenchymal enlargement (40.62% vs. 9.37%, *p*<0.0001), diffuse parenchymal hypoenhancement on both

phases (28.12% vs. 0%, $p=0.002$) and peripancreatic halo (34.37 vs. 0%, $p=0.0003$) were noted with higher frequency among AIP patients (Figure 2). The peripancreatic halo seen in AIP had a sensitivity and specificity of 0.34 and 1 respectively. Soft tissue infiltration around the superior mesenteric artery and vein were seen with a higher frequency in PA versus AIP patients (22% vs. 12%, $p=0.5$) (Figure 3). Non-cystic renal lesions were seen exclusively in patients with AIP (9%) (Figure 4). Findings are summarized in table 1.

Radiologist Accuracy

An accurate diagnosis of AIP was made by the 3 radiologists, in 23/32 (72%), 17/32 (53%) and 25/32 (78%) of cases, respectively, with a mean accuracy of 67.7%. An accurate diagnosis of PA was made by the 3 radiologists, in 25/32 (78%), 26/32 (81%) and 29/32 (91%) of controls, respectively, with a mean accuracy of 83.4%. The radiologists were uncertain in 2/32 (6%), 10/32 (31%) and 7/32 (22%) of cases respectively. Overall, the correct diagnosis was reached by at least two radiologists in 68.8% (22/32) of AIP cases and in 84.4% (27/32) of PA cases.

Most influential findings in the correctly diagnosed patients

This data was extracted separately from the free text responses provided by each radiologist regarding the presence or absence of the particular finding(s) from the provided list that helped them make the most likely diagnosis. The presence or absence of these features when agreed upon by 2 or more radiologists were then compared with the final clinical and pathologic diagnosis to assess if any of these particular findings played a part in making the correct or incorrect diagnosis. These numbers are different from the frequency of findings shown in table 1 as the presence or absence of a particular finding may or may not be regarded helpful in regards to making a particular diagnosis.

The diagnosis of AIP was made by 2 or more radiologists in a total of 27 cases. Among the 22 patients, in which at least 2 radiologists made the correct AIP diagnosis, diffuse parenchymal enlargement (68%), peripancreatic halo (55%), and diffuse parenchymal hypoenhancement (41%) in absence of all the following pattern including pancreatic ductal dilatation (95%), parenchymal atrophy (77%) or focal mass (77%) were found to be the most helpful findings in making this diagnosis.

The diagnosis of PA was made by 2 or more radiologists in a total of 37 cases. The presence of a focal mass (93%) followed by pancreatic ductal dilation (89%) were the leading findings that helped radiologists correctly identify PA.

Findings among Misclassified Patients

Among the 10 patients with a pathologic diagnosis of AIP, misdiagnosed as cancer by the radiologists, the most common finding was a focal mass in 90% (Figure 3). Pancreatic duct dilatation in 60% (Figure 5) and pancreatic parenchymal atrophy in 40% (Figure 6) were other findings that led to the diagnosis of cancer in patients with AIP.

The major reasons for the misclassification of 5 patients with pathological diagnosis of cancer as AIP were the absence of pancreatic atrophy (80%) followed by the presence of diffuse parenchymal enlargement (40%) (Figure 7) and peripancreatic halo (40%).

Interobserver Agreement

Using a three point scale, there was overall moderate agreement between radiologists in making a diagnosis of AIP or PA (κ : 0.58, $p < 0.0001$). Radiologists demonstrated near perfect agreement in identifying pancreatic calcifications (κ : 0.91, $p = 0.0003$) and the presence of a ductal stent (κ : 0.97, $p < 0.0001$).

Among the most influential CT findings that helped to make the diagnosis of AIP or PA, substantial agreement was reached for the identification of pancreatic duct dilatation (κ : 0.7, $p < 0.0001$) and parenchymal atrophy (κ : 0.67, $p < 0.0001$) while moderate agreement was reached for the identification of pancreatic mass (κ : 0.58, $p < 0.0001$) (Results summarized in Table 2).

Discussion

The diagnosis of AIP is usually based on a combination of imaging, serological, and histological findings recently classified by an international consensus conference.¹⁶ The response to corticosteroids also helps to confirm the diagnosis in the absence of characteristic imaging findings, histology, or serum IgG4 levels. However, the differentiation of AIP and pancreatic cancer can be challenging, especially in the presence of similar symptoms and signs as well as overlapping imaging findings. The reader accuracy varies depending upon the presence or absence of characteristic findings of these entities and overall experience of the individual radiologist.

AIP has been described as a diffusely enlarged hypoenhancing pancreas with a capsule like hypoattenuating rim or “halo”.¹¹ In our study, patients with AIP demonstrated diffuse pancreatic parenchymal enlargement, hypoenhancement and the characteristic peripancreatic halo with a significantly higher frequency than patients with PA. Peripancreatic halo, probably due to global inflammation of the pancreatic parenchyma and not due to extravasation of pancreatic juice, is a pattern of great value, especially when this halo is surrounding the entire gland. Similarly, patients with PA showed typical findings of a focal mass, pancreatic atrophy distal to the mass and pancreatic ductal dilation with a significantly higher frequency than patients with AIP. Pancreatic ductal enlargement of greater than 5 mm seen in PA has been used to differentiate from mass forming AIP.²⁵ However, overlapping findings including the presence of focal mass, pancreatic atrophy and pancreatic ductal dilatation in patients with AIP and the presence of diffuse parenchymal enlargement in PA were present. These findings often result in surgical resection. Common bile duct strictures were the most common extrapancreatic finding in patients with AIP, which is similar to previous studies.^{12, 17, 26, 27} In our study, common bile duct strictures were found with a similar incidence in AIP as patients with cancer. Due to presence of a stent in majority of these patients, further differentiation of benign and malignant features could not be made. Wall enhancement of the bile duct was appreciated in 15 patients (47%) with AIP, similar to previous reports,^{12, 21} compared to 7 patients (22%) with PA. However, in the

presence of a biliary stent, it is not clear if the enhancement was reactive due to the presence of the stent or related to the disease process of AIP. Extrapaneatic manifestations of AIP, including non-cystic renal lesions and retroperitoneal fibrosis, have been previously reported^{18, 19, 28} but were seen in only 3 and 2 patients, respectively. However, due to the rare occurrence of the extrapancreatic manifestations of AIP, statistical significance was not reached. The difference in perivascular involvement in patients with AIP and PA was not statistically significant. Inflammatory soft tissue stranding in AIP can mimic perivascular disease of PA. Soft tissue in the mesentery from sclerosing mesenteritis associated with AIP²⁹ can also sometimes mimic perivascular spread of pancreatic cancer as it did in one of our patients (Figure 3). Similarly, peripancreatic lymphadenopathy is a non-specific finding and was not different between the 2 entities. The radiologist accuracy in the present study was similar to a prior study where the accuracy of diagnosing PA was higher than AIP (84.4% vs. 67.7%) under similar conditions.¹² A higher reader accuracy of 93% for diagnosing AIP has also been reported previously.¹⁴ The difference in accuracy may be attributable to differences in patient inclusion criteria, as most of our patients underwent surgical resection due to the ambiguous clinical and imaging findings. Furthermore, all imaging studies were read by the radiologists independently and a consensus was not achieved on the final diagnosis. The accuracy also corresponded to the years of experience with the least experienced radiologists having the lowest accuracy. However, the number of equivocal responses provided by the same radiologist were also higher which may prompt further investigation to make the correct diagnosis.

A unique aspect of our study is an assessment of the key findings which helped the radiologists make the correct as well as the incorrect diagnosis of AIP and PA as this is a reflection of actual clinical practice and where opportunities might lie for imaging innovation. The radiologists found diffuse enlargement, peripancreatic halo and diffuse hypoenhancement along with the absence of typical findings seen in pancreatic cancer on CECT such as pancreatic ductal dilatation, focal mass and pancreatic atrophy most helpful in making the diagnosis of AIP. However, when presenting as a mass like lesion,^{30, 31} AIP can be difficult to differentiate from locally advanced pancreatic cancer. In our study, the presence of a focal mass was the leading reason for misdiagnosing AIP as PA. Similarly, the presence of parenchymal atrophy and ductal dilation, features that are commonly seen in PA but can occur in the presence of any obstructing mass in AIP, can mislead the radiologist towards misclassifying AIP as PA. Presence of parenchymal atrophy is due to complete stenosis of the MPD by PA leading to enlargement of the MPD upstream to the malignant stenosis. Atrophy in AIP is related to fibrous evolution of the disease, often without enlargement of the MPD. Some scenarios may pose a particular challenge to the radiologist in differentiating AIP from PA. For example, the presence of a CBD stent complicated the evaluation of duct wall enhancement and thickening, and often prevented stricture characterization (i.e., smooth and tapering versus abrupt) and introduce inflammation, features which may suggest AIP or raise its standing in the differential. The presence of the typical findings seen in PA were instrumental in making the diagnosis of PA with a substantial agreement among radiologists for the diagnosis of duct dilatation and parenchymal atrophy and moderate agreement in identification of a discrete pancreatic mass. In patients with PA that were incorrectly diagnosed as AIP, the absence of parenchymal

atrophy was the leading reason for the misdiagnosis. Pancreatic atrophy may be absent in patients with PA in the presence of a mass that is not obstructing the main duct. Findings such as diffuse parenchymal enlargement and peripancreatic halo may be seen due to superimposed acute pancreatitis because of obstructive pancreatitis and may have an appearance similar to AIP (Figure 7).

A similar study used a scale of 0–100% to indicate the confidence of the radiologist to distinguish AIP from PA.¹² Although this objectively indicates the confidence of the radiologist in making a diagnosis, the use of a 100 point scale is difficult to interpret for everyday clinical decision making. We used a 3 point scale in an attempt to increase the clinical usefulness of the radiologist's diagnosis since an abbreviated scale commits the radiologist to a diagnosis or an equivocal response. An equivocal response is important from the perspective of a radiologist since it will compel additional clinical, radiographic, and procedural evaluation necessary for elucidation of the correct diagnosis. Although this may potentially delay the surgery (possibly by 24–48 hours), a certain number of patients with AIP can be saved from the morbidity and mortality associated with a highly invasive surgery such as a pancreaticoduodenectomy.

Overall, the radiologists seemed to be best able to identify and agree upon findings related to changes in the pancreatic parenchyma such as enlargement or atrophy and the presence of pancreatic ductal dilatation as well as the presence of CBD wall enhancement. There was still moderate agreement in the detection of parenchymal hypoenhancement on both phases, a finding that may be subjective, and the presence of a peripancreatic halo which is a subtle finding. Moderate agreement was also seen for the detection of a focal mass lesion in both cancer and AIP patients. The extrapancreatic manifestations of AIP and PA were not as consistently identified with concordance. Although the low concordance rate could reflect the challenge of identifying often subtle findings of metastatic disease, it is expected our patient population would have a low incidence of these findings as metastatic disease usually precludes surgical resection. Renal lesions (Figure 4) are a common extrapancreatic finding in patients with AIP^{12, 18, 19, 32} and were recognized with moderate agreement among radiologists in the current study. However, the incidence of extrapancreatic manifestations likely varies based on the number of type 1 and type 2 AIP patients³³ and; therefore, the relative proportions of these AIP subtypes could result in varying degrees of interobserver agreement or disagreement. Regardless, it is important that a careful examination for extrapancreatic manifestations is undertaken in any patient with a suspicion for AIP as this could avert surgical resection. The major strengths of our study are that all patients were age and gender matched, most patients with AIP and all patients with PA had histological confirmation of disease. The lack of histologic confirmation in prior studies could have resulted in an overdiagnosis of AIP in their cohorts as the diagnostic criteria for AIP continue to evolve. Also, the imaging techniques between the two entities were similar as we matched each AIP case with a PA case with CECTs obtained in the same time period.

There are several limitations in the present study. Only 2 possible diagnoses were permissible. The presence of normal patients is usually desirable since it allows for an assessment of the negative predictive value of an abdominal imaging study. However, since most patients had positive findings, the diagnostic challenge for radiologists is usually to

differentiate between AIP and PA and the inclusion of a normal group would not answer our intended question. The option of an indeterminate diagnosis was provided in the questionnaire when the overall findings were equivocal to avoid committing to one of the 2 diagnoses. Another limitation of this study may be our inability to generalize our findings to community practice since radiologists at tertiary care hospitals have greater exposure to more uncommon diseases and may be able to differentiate between these two entities better than radiologists working in a community setting. However, in general, of all patients referred for presumed malignancy, only 20% patients are actual candidates for surgery⁷ as most patients are found to have locally advanced, unresectable disease. Approximately 10% of patients are found to have benign diseases, including focal chronic pancreatitis, pancreatic cysts and AIP. Masses more commonly form in type II AIP, as opposed to type I AIP, where 60% have masses and 40% have diffuse swelling.⁵ Since we did not have the data on the histologic subtypes of AIP, we could not assess their relative prevalence and how it might influence the interobserver agreement rates. A technical limitation of the study is the lack of uniformity of the imaging protocol including the amount and concentration of the contrast material and the section thickness as this data was acquired over a period of 12 years with changing imaging technology. In addition, a scan delay of 25–30 seconds was used for the arterial phase as opposed to the widely used pancreatic parenchymal phase of approximately 40 seconds. We used subjective evaluation of enhancement which is prone to perceptive error, however, the experience of the radiologists in assessment of normal enhancement pattern of pancreas was relied upon.

In conclusion, dual-phase CECT has reasonable accuracy for differentiating patients with AIP or PA. Abdominal radiologist must familiarize themselves with the pathognomonic CECT imaging manifestations of AIP such as diffuse pancreatic parenchymal enlargement, hypoenhancement and the characteristic peripancreatic halo in the absence of a focal mass, parenchymal atrophy distal to the mass and pancreatic ductal dilatation as these are all strong indicators for the diagnosis of AIP. However, an overlap of some of these findings, especially the presence of a focal mass, makes the diagnosis of AIP difficult and a search for other imaging findings such as the extrapancreatic manifestations may prove to be useful. When faced with a difficult or equivocal examination, further radiologic imaging with MRI/MRCP [26] and/or clinical evaluation (i.e., serum IgG4) or even tissue sampling (endoscopic ultrasound with core biopsy) should be considered.

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Figure 1.
a & b 63- year-old woman with autoimmune pancreatitis. Contrast enhanced axial CT scan demonstrated an enlarged pancreas with a surrounding peripancreatic halo (Fig 1a -white arrow) and intra- and extra-hepatic biliary ductal dilatation (Fig 1a -black arrow). Bile duct

wall enhancement is also present (Fig 1b-white arrow) in the absence of a pancreatic head mass. This was accurately diagnosed as autoimmune pancreatitis by all three radiologists.

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Figure 2.

58-year-old man with the history of type 1 autoimmune pancreatitis with a history of multiple extrapancreatic manifestations including renal cortical lesions, salivary gland swelling, sinus inflammation, and a distal bile duct stricture. Contrast-enhanced axial CT image demonstrates an enlarged hypo-enhancing pancreas. Extrapaneatic CBD dilatation due to a distal stricture along with wall enhancement due to cholangitis can also be seen (arrow). This was accurately diagnosed as autoimmune pancreatitis by all three radiologists.

Figure 3.

a&b: 65-year-old man with a history of autoimmune pancreatitis. Axial contrast enhanced CT shows a focal mass in the pancreatic tail (a-arrow). A soft tissue mass (b-thick arrow) is seen surrounding the superior mesenteric artery (b-thin arrow). The soft tissue mass was shown to be sclerosing mesenteritis on pathology. Slight hypoenhancement of the left kidney (Figure 3b) can also be appreciated which likely demonstrates renal involvement.

This was accurately diagnosed as autoimmune pancreatitis by one reader and was considered indeterminate and carcinoma by the other 2 radiologists respectively.

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Figure 4.

57-year-old man with autoimmune pancreatitis. Coronal contrast enhanced CT shows multiple hypoenhancing areas (arrows) in both kidneys consistent with renal involvement associated with AIP. This finding along with a hypoenhancing enlarged pancreas helped all three radiologists making the correct diagnosis.

Figure 5.

a&b: 80-year-old man with autoimmune pancreatitis. Axial contrast enhanced CT shows minimal pancreatic ductal dilatation (a-black arrow) with normal appearing pancreatic duct in the head region (b-thin arrow) and the presence of a very subtle hypodensity made all three radiologists lean towards cancer.

Figure 6.

a&b: 79-year-old woman with autoimmune pancreatitis. Axial contrast enhanced CT shows a mildly dilated duct (a-arrow) leading to a normal appearing pancreatic head without a mass lesion. There is pancreatic atrophy in the pancreatic tail (b-arrow). Intrahepatic biliary ductal dilatation is also seen (b). This was diagnosed as pancreatic cancer by all three radiologists.

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Figure 7.
a&b: 47-year-old man with pancreatic adenocarcinoma. Axial contrast enhanced CT shows pancreatic enlargement can be seen in the tail (a-arrow). Minimal peripancreatic stranding can also be seen compatible with acute pancreatitis. Hypodense mass can be seen in the pancreatic head (b-arrow). CBD stent also present (b-thin arrow). This was diagnosed as pancreatic adenocarcinoma by one reviewer, AIP by a second reviewer and indeterminate by the third reviewer.

Table 1

Frequency of imaging findings in autoimmune pancreatitis and PA.

Each of these findings was considered to be present when identified by at least 2 radiologists.

Findings:	Autoimmune pancreatitis	Pancreatic adenocarcinoma	p - value
Diffuse parenchymal enlargement	13 (41%)	3 (9%)	<0.0001
Diffuse parenchymal hypoenhancement	9 (28%)	0%	0.002
Peripancreatic halo or rim	11 (34%)	0 (0%)	0.0003
Retroperitoneal / para-aortic / paracaval stranding or fibrosis	2 (6%)	0 (0%)	0.49
Common bile duct stent	15 (47%)	17 (53%)	0.81
Common bile duct wall enhancement	15 (47%)	7 (22%)	0.06
Common bile duct stricture	20 (63%)	24 (75%)	0.28
Parenchymal calcifications	2 (6%)	2 (6%)	1
Focal mass	9 (28%)	25 (78%)	<0.0001
Parenchymal atrophy	3 (9%)	17 (53%)	<0.0001
Pancreatic ductal dilatation	3 (9%)	22 (69%)	<0.0001
Vascular involvement (Celiac axis, SMA, SMV)	4 (12%)	7 (22%)	0.51
Peripancreatic adenopathy	12 (38%)	14 (44%)	0.61
Non-cystic renal lesions	3 (9%)	0 (0%)	0.24

Table 2

Interobserver agreement for the diagnostic questions.

Findings	Kappa value	p value
Diffuse parenchymal enlargement	0.7	< 0.0001
Diffuse parenchymal hypoenhancement	0.43	0.006
Peripancreatic halo or rim	0.46	0.001
Retroperitoneal / para-aortic / pericaval stranding or fibrosis	0.58	0.05
Common bile duct stent	0.97	< 0.0001
Common bile duct wall enhancement	0.59	< 0.0001
Common bile duct stricture	0.29	< 0.0001
Parenchymal calcifications	0.91	0.0003
Absence of focal mass	0.58	< 0.0001
Absence of parenchymal atrophy	0.67	< 0.0001
Absence of pancreatic ductal dilatation	0.7	< 0.0001
Vascular involvement (Celiac axis, SMA, SMV)	0.37	0.007
Peripancreatic adenopathy	0.08	0.28
Non-cystic renal lesions	0.5	0.05