

Simulators of Pancreatic Adenocarcinoma: Key Findings to Avoid Misdiagnosis

Atanas Dimitrov Hilendarov*, Alexandar Asenov Georgiev and Lyubomir Georgiev Chervenkov

Department of Diagnostic Imaging, Faculty of Medicine, Medical University, Plovdiv, Bulgaria

*Corresponding author:

Atanas Dimitrov Hilendarov,
Department of Diagnostic Imaging, Faculty of
Medicine, Medical University, Plovdiv, Bulgaria

Received: 22 Sep 2026

Accepted: 11 Oct 2026

Published: 14 Oct 2026

J Short Name: ACMCR

Copyright:

©2026 Atanas Dimitrov Hilendarov.

This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially

Citation:

Atanas Dimitrov Hilendarov, Simulators of Pancreatic Adenocarcinoma: Key Findings to Avoid Misdiagnosis. Ann Clin Med Case Rep® 2026; V17(1): 1-7

1. Abstract

1.1. Purpose

To present examination finding of neoplastic, inflammatory and benign pancreatic as well as non-pancreatic processes that can simulate pancreatic adenocarcinoma (PA).

2. Introduction

Adenocarcinoma of the pancreas (PA) is one of the most aggressive malignancies, the fourth leading cause of cancer-related deaths in the Western world. Timely and accurate diagnosis of PA is critical, as the disease has a high incidence and high mortality despite treatment measures. Tumor markers, mainly CA 19-9, and biopsy under US or CT control can help make the correct diagnosis, but they also have several drawbacks. While CA 19-9 is elevated in 81% of PA cases, although in its absence it cannot be used to rule out malignancies, its negative prognostic value is very low (69%). EUS-FNA and CT-guided fine-needle biopsy have high accuracy but low sensitivity in diagnosing PA. EUS-FNA has a limited role in cases of duodenal stenosis and in lesions that are located in the uncinate process or in the tail, and CT-guided wide-needle biopsy has a limited role in small lesions. Both biopsy techniques cannot be performed in cases of blood vessel interposition.

These are the reasons why the guidelines indicate that when there is a high suspicion of malignancy of a solid lesion in the pancreas, histological evidence of PA is not a reason for pancreatectomy. This fact inevitably led to a high percentage of false positive pancreatectomies; 5% to 10% of pancreatectomies performed with a clinical diagnosis of PA turn out to be pseudotumors. Making an accurate diagnosis of PA with imaging is crucial to prevent unnecessary surgical procedures [5,6,7,8]. Many benign, congenital, chronic inflammatory and neoplastic diseases can mimic PA and are likely to be a challenge for specialist radiologists or more experienced radiologists dealing with abdominal examinations [1,2,3,4]. The purpose of this presentation is to provide the key criteria that presuppose the correct

diagnosis, mainly through the assessment of CT, which is the preferred method for imaging the pancreas, which is also our object of interest.

3. Imaging Findings and Details of The Selected Cases

3.1. Focal Fatty Infiltration

Fatty infiltration of the pancreas can be focal or diffuse. This is a benign change in the structure of the pancreas associated with diabetes, obesity, focal pancreatitis, steroid therapy and others conditions. Focal fatty infiltration is usually most pronounced in the head of the pancreas and at contrast enhancement in CT usually appears as a region with reduced contrast material amplification compared to normal pancreatic tissue simulating PA. The key finding that leads to a correct diagnosis is the presence of a fat density lesion in a non-contrast CT scan or a decrease in the signal in the opposite phase chemical shift in MRI. Additional findings in favour of focal fatty infiltration are - no enlargement of the pancreas or dilatation of the pancreatic duct and bile ducts - the lack of coverage of neighboring vessels, and the absence of lymphadenopathy Figure 1.

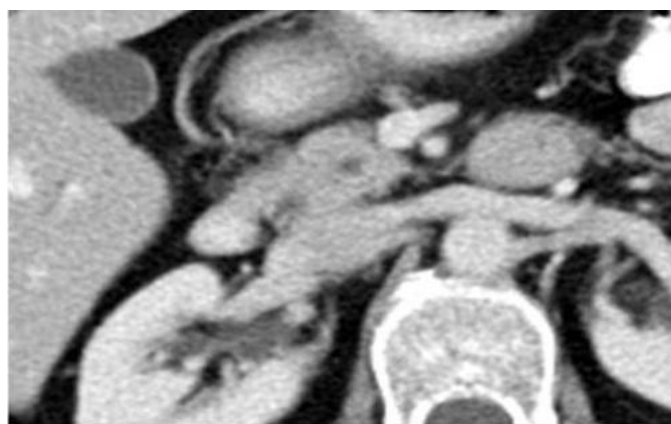


Figure 1: CT of the pancreas- portal phase: Focal fatty infiltration.

3.2. This Pancreatic Cancer?

3.2.1. Hyperintensive Lesion in The Head of The Pancreas

3.2.1.1. Diverticulum of the Duodenum

The duodenum is the second most common area of the gastrointestinal tract after the colon, where diverticula usually form in the peripancreatic region. They are usually present with a hydroaerial level or with the presence of an oral contrast agent in

the lumen. However, sometimes diverticula are misdiagnosed as pancreatic tumors. The collapsed one a diverticulum or one filled with fluid may become a suspicious lesion of the pancreas. Cases of diverticulitis or rupture of diverticula are even more difficult to diagnose correctly [14,17]. In such cases, endoscopy can contribute to a correct diagnosis. Alternatively, CT can be repeated within a few days after calming of clinical and radiological findings Figure 2.

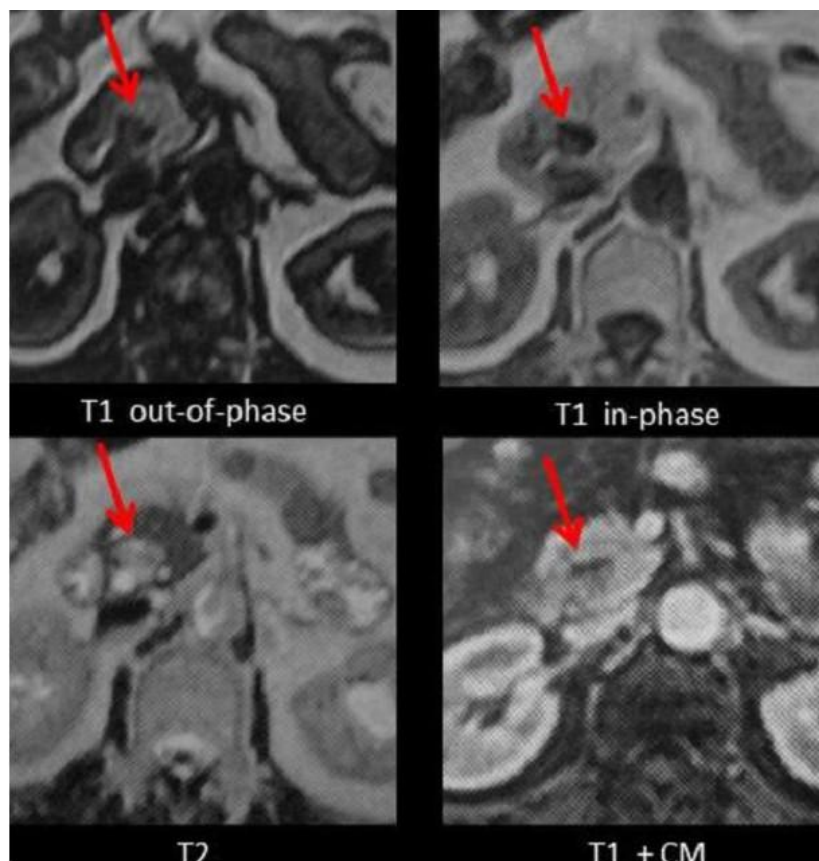


Figure 2: MRI of the pancreas: Duodenal diverticulum.

4. Is It Pancreatic Cancer?

4.1. Exophytic Formation in The Head of The Pancreas

4.1.1. Normal Variations in The Contour of The Pancreatic Head

Normal variations in the contour of the pancreatic head can sometimes mimic malignancy. When normal pancreatic tissue extends over 1 cm relative to the anterior superior pancreaticoduodenal artery (ASPD), it is considered as lobulation of the head of the pancreas. There are three types of parenchymal bulges: Types I, II and III refer to anterior, posterior and horizontal bulges relative to ASPD, respectively. Knowledge of these changes is important to overcome their misinterpretation, such as PA. The main finding, which proves the presence of normal variation of the pancreatic contour, is that this swelling has similar structures and behavior with the rest of the pancreatic parenchyma in all phases (non-contrast, pancreatic, portal and venous phases) [23,24,25]. Coronal and sagittal image reformats can provide additional help in visualizing the lobular contour of the gland Figure 3.

5. CT Images

5.1. Hypoattenuated Lesion of The Head of The Pancreas

5.1.1. Annular Pancreas

The annular pancreas is a rare variant in which the pancreatic parenchyma bypasses the second part of the duodenum in whole or in part. When it is symptomatic in children, it usually presents with symptoms of stomach obstruction. In adults, this is often an accidental finding or occurs with symptoms of obstruction, peptic ulcers or pancreatitis. When the intrapancreatic portion of the descending duodenum is not clearly contrasted, a falsely suspicious lesion may be admitted [4,5]. Key finding in favour of the annular pancreas is the discovery of a Santorini canal in normal pancreatic tissue that surrounds the duodenum. In some cases, stenosis of the descending duodenum may be depicted. In uncertain cases, secretin contrast MRI can help us make the correct diagnosis Figure 4.

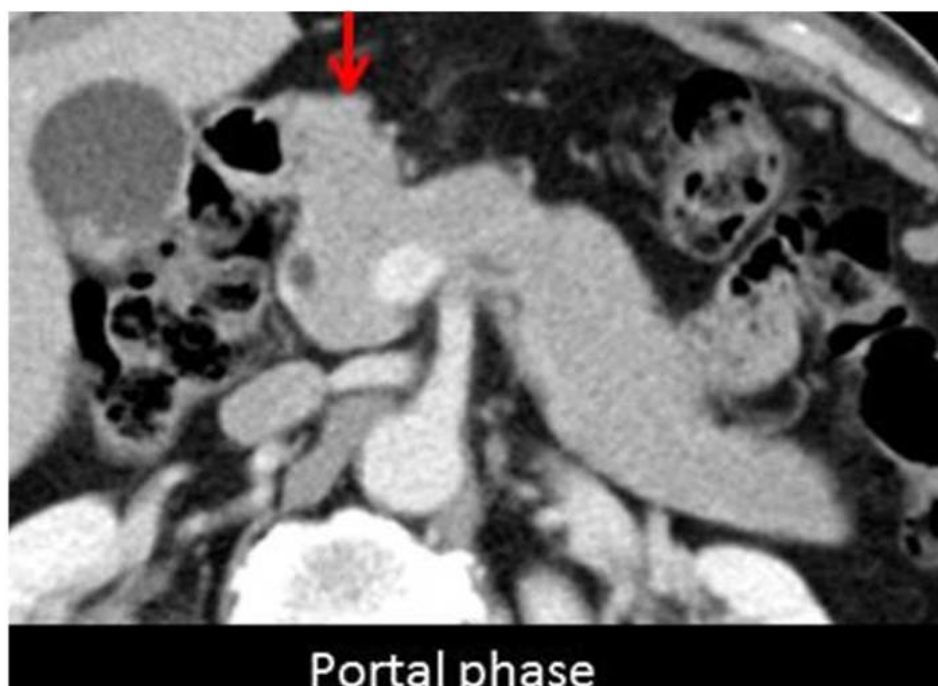


Figure 3: CT images - Normal version of the contour of the pancreatic head.

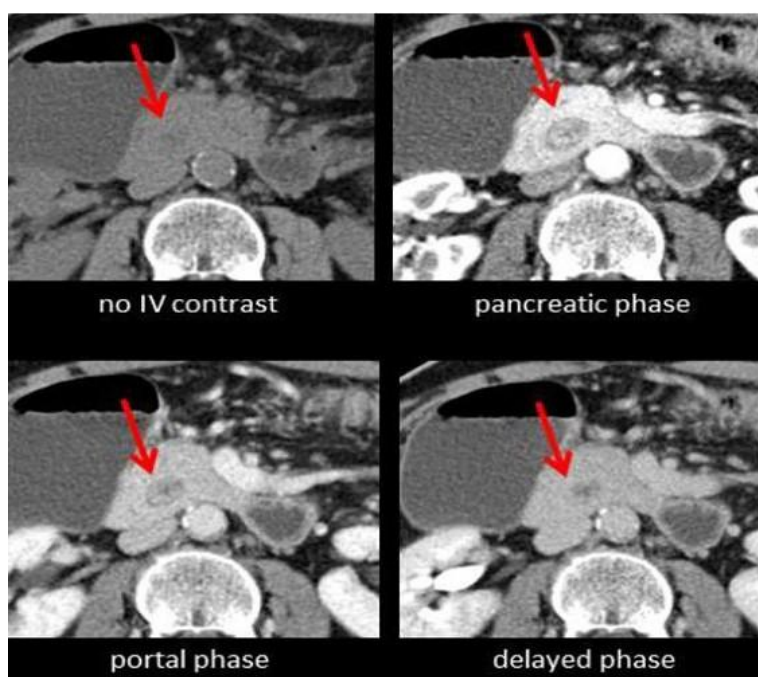


Figure 4: CT images: Annular pancreas.

6. Is This a Pancreatic Cancer Is on This Image?

6.1. CT Image With Contrast Enhancement in Inhomogeneous Mass in The Head of The Pancreas

6.1.1. Focal Chronic Pancreatitis

On imaging, chronic pancreatitis is characterized by parenchymal atrophy, dilatation of the main pancreatic duct, and the presence of calcification. Nearly 20% of chronic cases of pancreatitis develop a focal form that can simulate PA and is usually associated with increased alcohol consumption. In CT, focal chronic pancreatitis manifests itself as a low-density mass that causes peripheral dilatation of the main pancreatic duct and atrophy of

the peripheral pancreas parenchyma. Imaging of a penetrating pancreatic duct in a suspicious the lesion is indicative of an inflammatory pseudotumor as well as the absence of involvement of the vessels in the peripancreatic zone. Diffusion-weighted MRI provides additional help in ambiguous cases, as it shows a lower diffusion coefficient (ADC) in PA compared to the rest of the pancreatic tissue, but no significant differences from the ADC values in focal chronic pancreatitis with the rest of the pancreas. (4,10,14). However, according to the guidelines, in patients with chronic pancreatitis and increased resemblance to PA, resection is recommended even without histological evidence Figure 5.

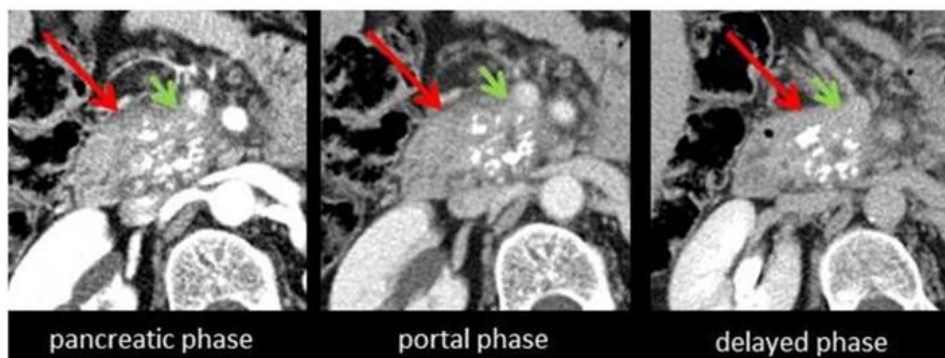


Figure 5: CT images - Focal chronic pancreatitis.

7. Pancreatic Mass with Poor Contrast Saturation.

7.1. Is This a Pancreatic Cancer?

7.1.1. Paraduodenal Pancreatitis

Paraduodenal pancreatitis (PP) is another separate form of chronic pancreatitis critically discussed. Although the exact cause is not known, it is usually presented in middle-aged men with a history of alcohol consumption with persistent (3-6 months) episodes of jaundice and weight loss or with symptoms of acute pancreatitis. It is divided into two types: inflammatory tissue and fibrous change can affect either only the (clean) region of the groove or cover the head of the pancreas (segmental). On CT, the pure form usually appears as a parenchymal mass of soft tissue in the groove area, which is described as leaf-like tissue

that shows late saturation due to fibrosis. Additional saturation of the pancreatic head occurs in the segmental form. Both types can mimic PA and, unfortunately, even negative FNA results cannot rule out malignancy with absolute certainty. The specific location of the PP is an important indication for diagnosis, as is the displacement of the vascular system in the absence of obstruction. The CT characteristics of PP are a thickening of the duodenal wall and a slight heterogeneous saturation other than PA, which usually contrasts homogeneously.(4,14). In addition, the smooth, prolonged narrowing of the pancreas and common bile duct favors the diagnosis of PP. Conversely, pronounced dilatation of the pancreatic ducts or abrupt rupture of the pancreatic duct are suspicious data indicating the presence of malignancy/ Fig.6/.

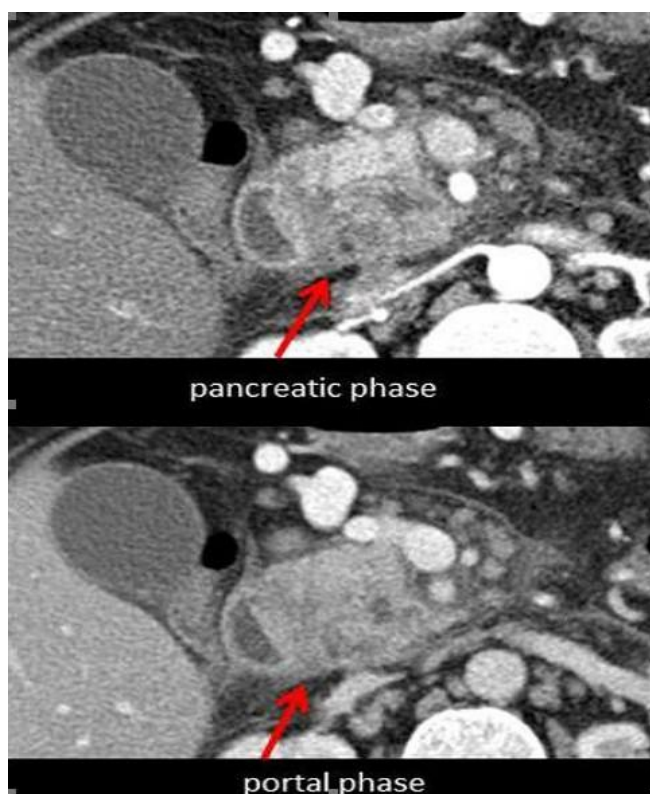


Figure 6: CT images - Paraduodenal pancreatitis.

8. Inhomogeneous Mass with Poor Saturation in The Head of The Pancreas.

8.1. Pancreatic cancer?

8.1.1. Autoimmune Pancreatitis

Autoimmune pancreatitis (AIP) is one of the most common misdiagnosis that leads to suspected pancreatic surgery. This is a different type of chronic pancreatitis, which is impressively affected by steroids and is divided into two types. Type 1 AIP is IgG4 associated with extra-pancreatic manifestations (sclerosing mesenteritis, Sjogrenlike syndrome, retroperitoneal fibrosis, interstitial nephritis), while AIP in Type 2 coexists with IBD in 30% of cases. Diffuse enlargement of the sausage-like parenchyma with obliteration of the lobular contour of the pancreas and capsule-like contouring with delayed contrast are typical radiological signs of autoimmune pancreatitis [18,19]. The findings of the image suggesting a focal AIP are appreciable inhomogeneous enhancement in the pancreatic phase and homogeneous improvement in the late phase, in contrast to PA, which shows a reduced magnification in both phases. Another differential feature with high AIP specificity is the “capsule-like” low saturation edge as opposed to the high contrast frame that surrounds PA in some cases. In addition, the segmental narrowing of the main pancreatic duct (MPD) is usually multifocal and milder in AIP, while dilatation of the MPD is more than 5 mm distal to the lesion, suggesting malignancy. The high suspicion of AIP should be confirmed by high serum IgG4 levels and by EU-targeted biopsy. In case of ambiguous biopsy results, 4-6 weeks of steroid treatment is administered and recommended for therapeutic and diagnostic purposes Figure 7.

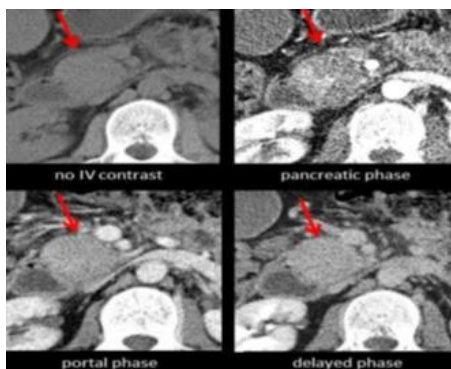


Figure 7: CT images in different phases-Autoimmune pancreatitis.

9. Pancreatic Neuroendocrine Tumors

Pancreatic neuroendocrine tumors (PNETs) originate from well-differentiated endocrine pancreatic cells and are composed of proliferations ranging from benign to malignant of 5-10% of pancreatic tumors. PNETs are usually presented as dense masses that are displayed in the arterial phase. Unlike PA, they are well labeled and do not cause dilatation of the pancreatic ducts, with the exception of small serotonin-secreting tumors, which tend to cause obstruction of the pancreatic duct due to the formation of fibrous tissue. In challenging cases with heterogeneous morphology or diagnosis of pancreatic steroid scintigraphy can help us make the correct diagnosis. In PNET, liver metastases are

not uncommon and are usually hypervascular [8,9,11]. They are small and necrotic or hemorrhagic when larger. Although liver metastases from PA are an absolute contraindication to surgery when it comes to PNET metastatic liver disease, the behavior is far different. Patients with PNET and liver metastases appear to benefit from primary resection and multimodal therapy of liver metastases Figure 8.

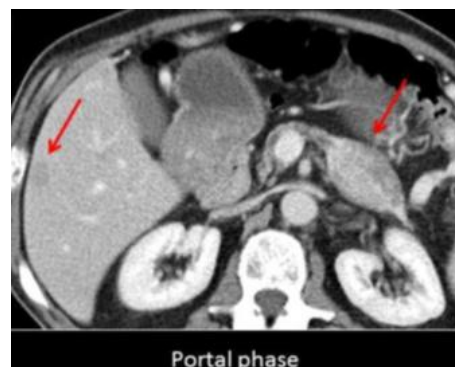


Figure 8: CT image in the portal phase-Pancreatic neuroendocrine tumor with liver metastases.

10. Lymphomas

Secondary involvement of the pancreas by lymphoma (mainly non-Hodgkin's) is not uncommon and it is often part of the extranodal involvement in 5-30% of patients. This can occur either by infiltration from adjacent peripancreatic lymph nodes or by hematogenous spread. Primary lymphoma of the pancreas is quite rare. The focal form of pancreatic lymphoma usually presents as a single, voluminous, homogeneous lesion with lower muscle attenuation and shows mild homogeneous saturation in contrast to enhanced CT [15,16]. Knowing the patient's history of lymphoma when secondary pancreatic lesions are detected and the presence of a bulk mass that does not cause significant dilatation of the pancreatic ducts makes the diagnosis of lymphoma more favourable than PA. In addition, lymphomatous involvement of the pancreas can cause a lining of the food vessel without invasion. Finally, the presence of enlarged lymph nodes below the level of the renal veins is another important image in favour of lymphoma figure 9.

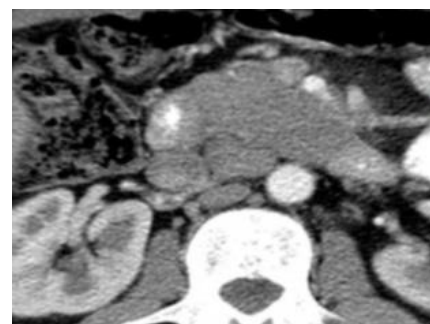


Figure 9: CT image- Lymphoma.

11. Is This a Pancreatic Cancer?

11.1. Metastases

Pancreatic metastases are quite rare and represent 1-2% of all pancreatic neoplasms. The most common primary tumors that can metastasize to the pancreas are kidney tumor, lung cancer,

breast cancer, melanoma, colon cancer, stomach cancer, ovarian cancer, and prostate cancer. Metastases are often multifocal and usually well defined, in contrast to PA, which is usually solitary and tends to have unclear boundaries. In most cases of pancreatic metastases, the main pancreatic duct and the common bile duct are not dilated compared to PA [13,14,15]. Must be noted that hypercontrast lesions typically originate from a hypervascular neoplasm, such as renal cancer, and hypocontrast lesions originate from hypovascular tumors, such as lung cancer Figure 10.

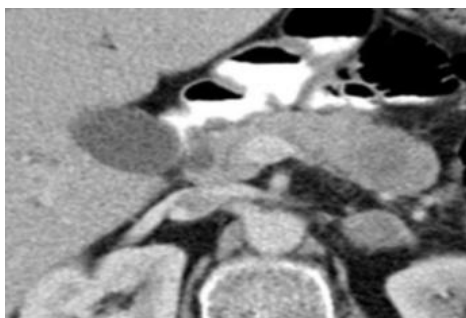


Figure 10: CT images-Metastases.

12. Conclusion

Imaging of a hypoattenuating lesion in the head of the pancreas corresponds to adenocarcinoma in most cases. However, radiologists should be careful, because a false positive diagnosis can lead to unnecessary surgery, which is not without complications. Proper diagnosis of benign conditions or other types of malignancies beyond PA with significantly better survival is important because it suggests alternative therapeutic approaches [6,7,8]. Radiologists should be familiar with these imitators of pancreatic adenocarcinoma to make the correct diagnosis and to assist the physician in favour of the patient.

References

1. Al-Hawary MM, Kaza RK, Azar SF, Ruma JA, Francis IR. Mimics of pancreatic ductal adenocarcinoma. *Cancer Imaging*. 2013; 13(3): 342-349.
2. Schima W, Ba-Ssalamah A, Kölblinger C. Pancreatic adenocarcinoma. *Eur Radiol*. 2007; 17: 638.
3. Zamboni G, Capelli P, Scarpa A, Bogina G, Pesci A, Brunello E, Klöppel G. Nonneoplastic mimickers of pancreatic neoplasms. *Arch Pathol Lab Med*. 2009; 133: 439-453.
4. Okun SD, Lewin DN. Non-neoplastic pancreatic lesions that may mimic malignancy. *Semin Diagn Pathol*. 2016; 33: 31-34.
5. Lermite E, Sommacale D, Piardi T. Complications after pancreatic resection: diagnosis, prevention and management. *Clin Res Hepatol Gastroenterol*. 2013; 37: 230-239.
6. Keim V, Klar E, Poll M, Schoenberg MH. Postoperative Care Following Pancreatic Surgery: Surveillance and Treatment. *Deutsches Ärzteblatt International*. 2009; 106(48): 789-794.
7. Ho C-K, Kleeff J, Friess H and Buchler MW. Complications of pancreatic surgery. *HPB*. 2005; 7: 99-108.
8. Nguyen SQ, Angel LP, Divino CM, Schluender S and Warner RRP. Surgery in malignant pancreatic neuroendocrine tumors. *J Surg Oncol*. 2007; 96: 397-403.
9. Iglesias-García J, Lindkvist B, Lariño-Noia J. The role of EUS in relation to other imaging modalities in the differential diagnosis between mass forming chronic pancreatitis, autoimmune pancreatitis and ductal pancreatic adenocarcinoma. *Rev Esp Enferm Dig*. 2012; 104: 315-321.
10. Tyng CJ, Almeida MFA, Barbosa PN. Computed tomography-guided percutaneous core needle biopsy in pancreatic tumor diagnosis. *World Journal of Gastroenterology*. 2015; 21(12): 3579-3586.
11. Raman SP, Hruban RH, Cameron JL, et al. Pancreatic imaging mimics: part 2, pancreatic neuroendocrine tumors and their mimics. *AJR Am J Roentgenol*. 2012; 199: 309-318.
12. Coakley FV, Hanley-Knutson K, Mongan J, Barajas R, Bucknor M, Qayyum A. Pancreatic imaging mimics: part 1, imaging mimics of pancreatic adenocarcinoma. *AJR Am J Roentgenol*. 2012; 199(2): 301-308.
13. Triantopoulou C, Kolliakou E, Karoumpalis I, Yarmenitis S, Dervenis C. Metastatic disease to the pancreas: an imaging challenge. *Insights into Imaging*. 2012; 3(2): 165-172.
14. Kaza RK, Al-Hawary MM, Sokhandon F, Shirkhoda A, Francis IR. Pitfalls in pancreatic imaging. *Seminars in Roentgenology*. 2015; 50(4): 320-327.
15. Alzahrani MA, Schmulewitz N, Grewal S, Lucas FV, Turner KO. Metastases to the pancreas: the experience of a high volume center and a review of the literature. *J Surg Oncol*. 2012; 105: 156-161.
16. Merkle EM, Bender GN, Brambs HJ. Imaging findings in pancreatic lymphoma: differential aspects. *AJR Am J Roentgenol*. 2000; 174: 671-5.
17. Macari M, Lazarus D, Israel G, Megibow A. Duodenal diverticula mimicking cystic neoplasms of the pancreas: CT and MR imaging findings in seven patients. *AJR Am J Roentgenol*. 2003; 180: 195-199.
18. Crosara S, D'Onofrio M, De Robertis R. Autoimmune pancreatitis: Multimodality non-invasive imaging diagnosis. *World Journal of Gastroenterology*. 2014; 20(45): 16881-16890.
19. Zaheer A, Singh VK, Akshintala VS. Differentiating Autoimmune Pancreatitis from Pancreatic Adenocarcinoma using Dual-phase Computed Tomography: An Inter-observer Study. *Journal of computer assisted tomography*. 2014; 38(1): 146-152.
20. Asbun HJ, Conlon K, Fernandez-Cruz L, et al. When to perform a pancreatoduodenectomy in the absence of positive histology? a consensus statement by the international study group of pancreatic surgery. *Surgery* 2014; 155(5): 887-892.
21. Yu J, Turner MA, Fulcher AS, Halvorsen RA. Congenital anomalies and normal variants of the pancreaticobiliary tract and the pancreas in adults: part 2. Pancreatic duct and pancreas. *Am J Roentgenol*. 2006; 187: 1544-53.
22. Gandhi NS, Feldman MK, Le O, et al. Imaging mimics of pancreatic ductal adenocarcinoma. *Abdom Radiol*. 2017.
23. Fattahi R, Balci NC, Perman WH, Hsueh EC, Alkaade S. Pancreatic diffusion-weighted imaging (DWI): comparison between mass-forming focal pancreatitis (FP), pancreatic cancer (PC), and normal pancreas. *J Magn Reson Imaging*. 2009; 29: 350-356.

24. Lee JE, Shin SS, Kim SJ, Heo SH, Lim HS, Kim JW, Jeong YY. A pictorial review of diagnostic pitfalls of developmental anomalies and variants in pancreatic imaging. *Clin Imaging* 2017; 48: 32-39.
25. Borghei P, Sokhandon F, Shirkhoda A. Anomalies, anatomic variants, and sources of diagnostic pitfalls in pancreatic imaging. *Radiology*. 2013; 266: 28-36.