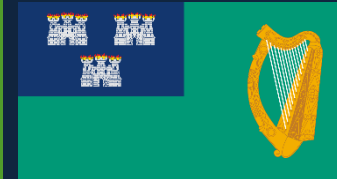


Pancreatic masses with calcifications and cystic/necrotic changes (**dual morphology**)

How do I interpret the scan correctly?

**ID 53817 - Binit Sureka
EE-163**



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Compliance with ethical standards

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Learning Objectives

1. Identify all the lesions (inflammatory, developmental and neoplastic) in pancreas that can show both calcification and cystic/necrotic changes
2. Discuss the key imaging features of these lesions based on the morphology
3. To tabulate diagnostic approach to pancreatic masses with both calcifications and cystic/necrotic changes

Background

- Pancreatic calcifications with cystic/necrotic changes are key features commonly used to diagnose various types of pancreatic disease.
- Chronic calcific pancreatitis secondary to alcohol use is the most common cause of pancreatic calcifications.
- Other pathologic conditions (developmental, neoplasm) also can cause calcifications.
- Awareness of these entities and their classic features is important in making the correct diagnosis and guiding proper management.
- In this presentation, we discuss various causes of pancreatic calcifications with cystic/necrotic changes and their imaging appearances at CT/MRI.

Differential Diagnosis of Pancreatic calcifications with cystic/necrotic changes

Pathological Entity	Pattern of calcification	Remarks	Incidence (%)
Pseudocyst	Outer wall ; milk of calcium	Additional features of pancreatitis	Rare
Serous cystic neoplasm	Central stellate scar	Grandmother lesion (5 th -6 th decade)	30
Mucinous cystic neoplasm	Curvilinear/punctate	Mother lesion (4 th -5 th decade)	15
IPMN	Intraductal calcification	Grandfather lesion (6 th -7 th decade); absence of features of pancreatitis	Rare
SPEN	Peripheral punctate	Daughter lesion; young females	30
Adenocarcinoma	Adjacent to mass	Hypo enhancing; MPD dilatation; vascular invasion; elevated CA19.9	Rare

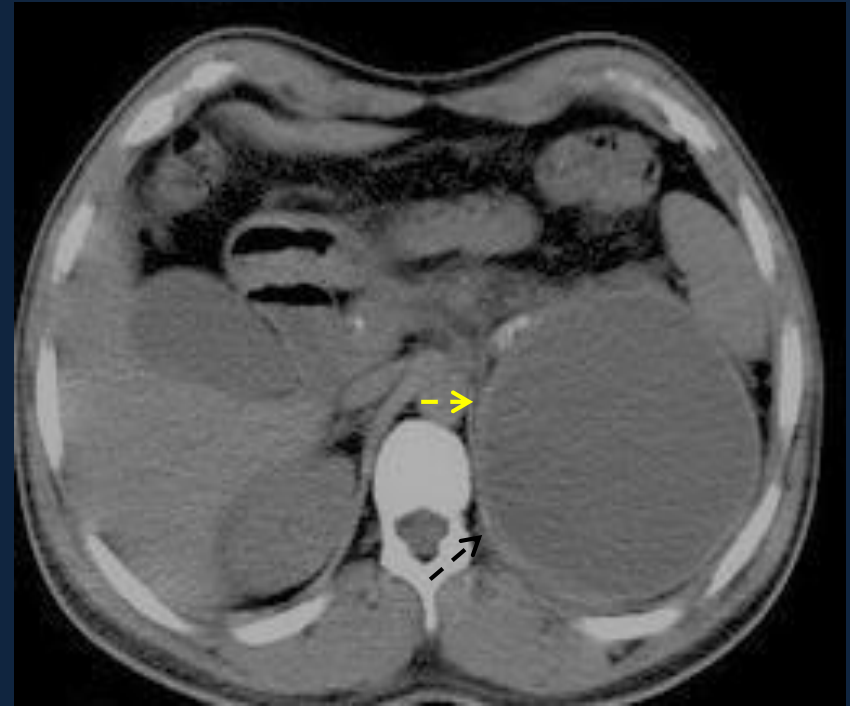
Other rare causes are infectious cysts, parasitic cysts, cystic teratoma, genetic mutation associated pancreatitis

Differential Diagnosis of Pancreatic calcifications with cystic/necrotic changes

Pathological Entity	Pattern of calcification	Remarks	Incidence (%)
Focal mass forming pancreatitis	Parenchymal, Intraductal	Features of pancreatitis	50
Neuroendocrine tumor	Focal, coarse calcification	Seen in nonfunctioning NETs	22
Acinar cell carcinoma	Variable, nonspecific	Seen in elderly females	6-50
Calcified metastasis	Variable, nonspecific	Metastasis to pancreas is very rare	Rare
Other causes: Calculus Vascular	Intraductal calculus Wall of pseudoaneurysm	Associated with chronic pancreatitis Associated with atherosclerosis	Variable
Other rare causes are infectious cysts, parasitic cysts, cystic teratoma, genetic mutation associated pancreatitis			

Pseudocyst

- ✓ H/o pancreatitis
- ✓ H/o alcohol, stone disease, abdominal trauma
- ✓ Unilocular
- ✓ Non-enhancing dependent debris
- ✓ Milk of calcium is due to a fluid-calcium level in pancreatic pseudocysts.
- ✓ The cause of milk of calcium is unclear. It has been suggested, however, that a pseudocyst causes stasis of calcium-containing suspensions, which results in dependent layering of calcium and is seen as high-attenuation material on CT images



46/M: Axial CT image showing pancreatic pseudocyst with *subtle peripheral curvilinear calcification* (arrow) in the wall of the pseudocyst

Serous cystic neoplasm

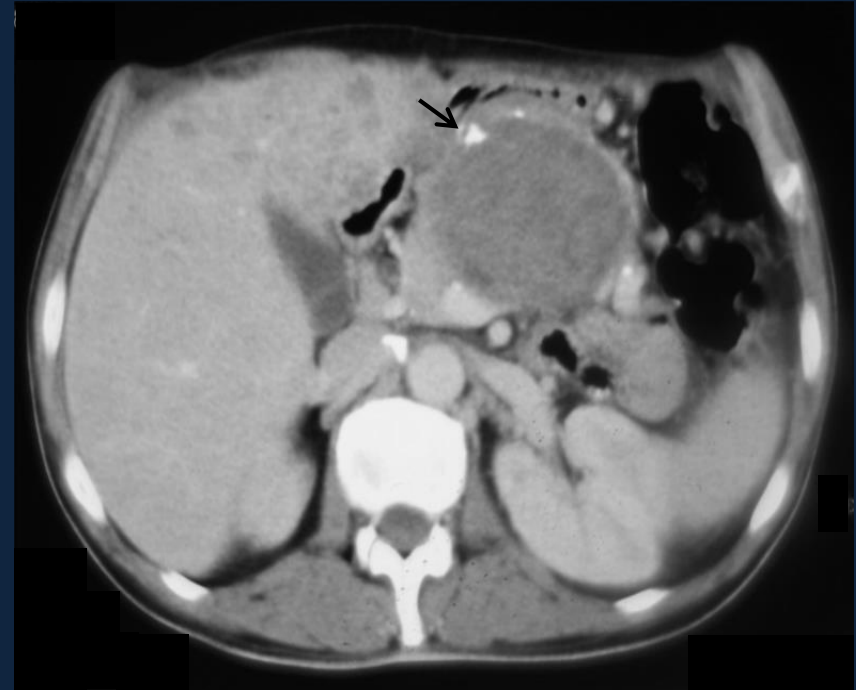
- ✓ 'Grandma' (6-7th decade) [F:M=3:1]
- ✓ Benign tumor
- ✓ Lobulated, microcystic (>6, each <2cm), thin wall
- ✓ Central scar (30%) and calcifications (18%)
- ✓ Macrocytic – 10%, polycystic and oligocystic (<10%)
- ✓ Rarely hypervascular enhancement
- ✓ Malignant transformation is extremely rare



63-year-old-female: Axial CECT image showing polycystic mass lesion in head of pancreas with **central stellate calcification** in a case of serous cystic neoplasm of pancreas

Mucinous cystic neoplasm

- ✓ Exclusively seen in women - 'Mother' (4-5th decade)
- ✓ M:F ratio <1:20
- ✓ Macrocytic (<6, each >2cm), thick wall
- ✓ Septations, peripheral calcification (15%)
- ✓ Location in the tail and body of the pancreas (95%)
- ✓ Premalignant (solid, enhancing, calcification, irregular wall)



48-year-old-male: Axial CECT image showing solid-cystic mass lesion in tail of pancreas *with punctate curvilinear calcification* in periphery of the lesion in a case of mucinous cystadenocarcinoma

Focal mass forming pancreatitis

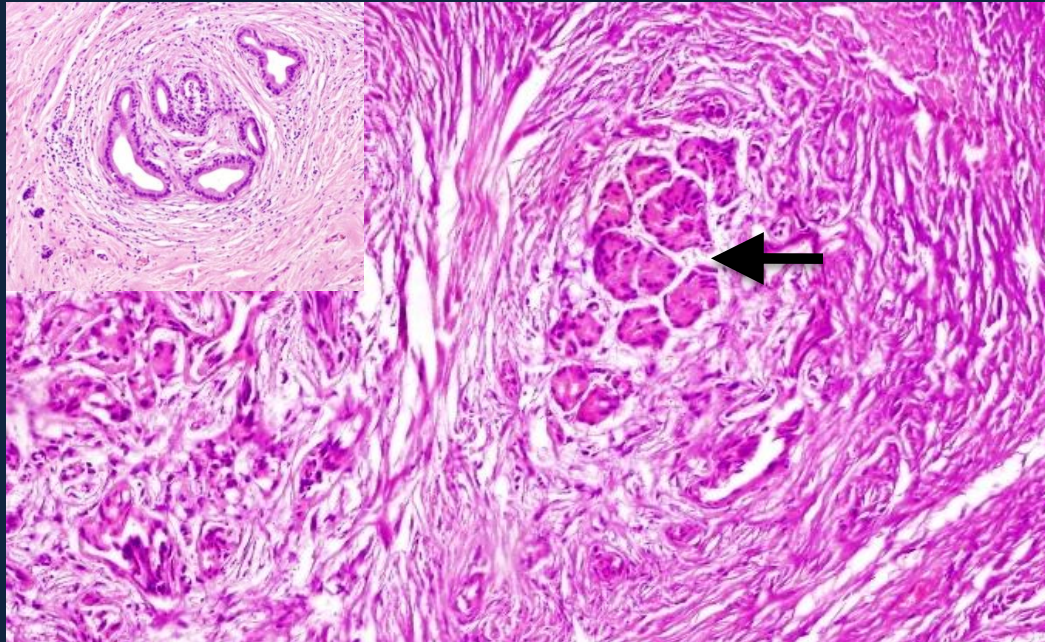
- ✓ The risk of developing pancreatic cancer in patients with chronic pancreatitis is about 15 times higher than in the average population
- ✓ Clinical features and biochemical parameters that suggest malignant mass in head of pancreas are older age, persistent jaundice, worsening abdominal pain, gastric outlet obstruction, significant weight loss and elevated CA 19:9 greater than 300 U/mL
- ✓ Conventional CT however may have difficulty in differentiating between inflammatory and neoplastic masses as well as detecting lesions < 2 cm in diameter as small tumors are sometimes isoattenuating to background pancreatic parenchyma.
- ✓ MRI: Assessment of pancreatic ductal structures can sometimes provide a clue as pancreatic cancers may lack pancreatic ductal structures while a pseudotumour may contain dilated side branches
- ✓ Choi et al have shown that multiplicity, similar or high signal intensity on portal phase and 3- and 20-minute delayed phase images, homogeneous enhancement, no peripancreatic fat infiltration, no internal cystic or necrotic portion, capsulelike rim, no upstream pancreatitis, no vascular invasion, and duct penetrating sign were more frequently observed ($p < 0.05$) in mass-forming AIP.
- ✓ The apparent diffusion coefficient (ADC) value was also significantly lower for mass-forming AIP than for PDAC

Focal mass forming pancreatitis



51-year-old-male: Axial CECT image showing inflammatory mass in head of pancreas mimicking malignancy with *intraductal calculi* in side branches in a case of mass forming pancreatitis

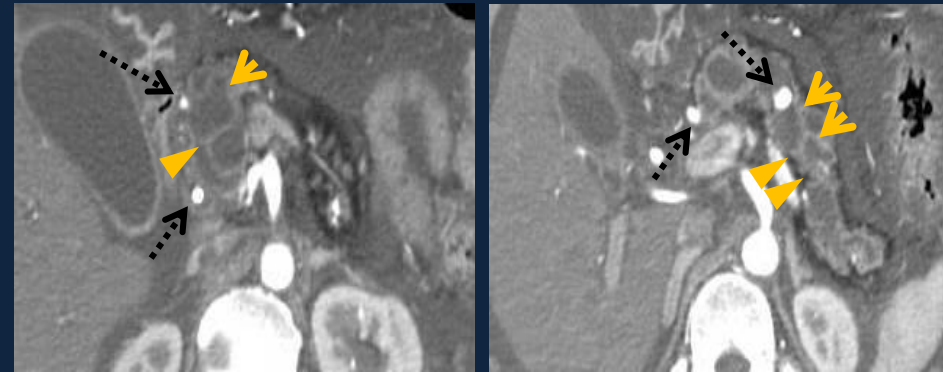
Chronic pancreatitis



Histopathology shows fibrotic destruction of pancreatic parenchyma and atrophic acini, periductal fibrosis with variable dilatation and obstruction of pancreatic ducts (Inset)

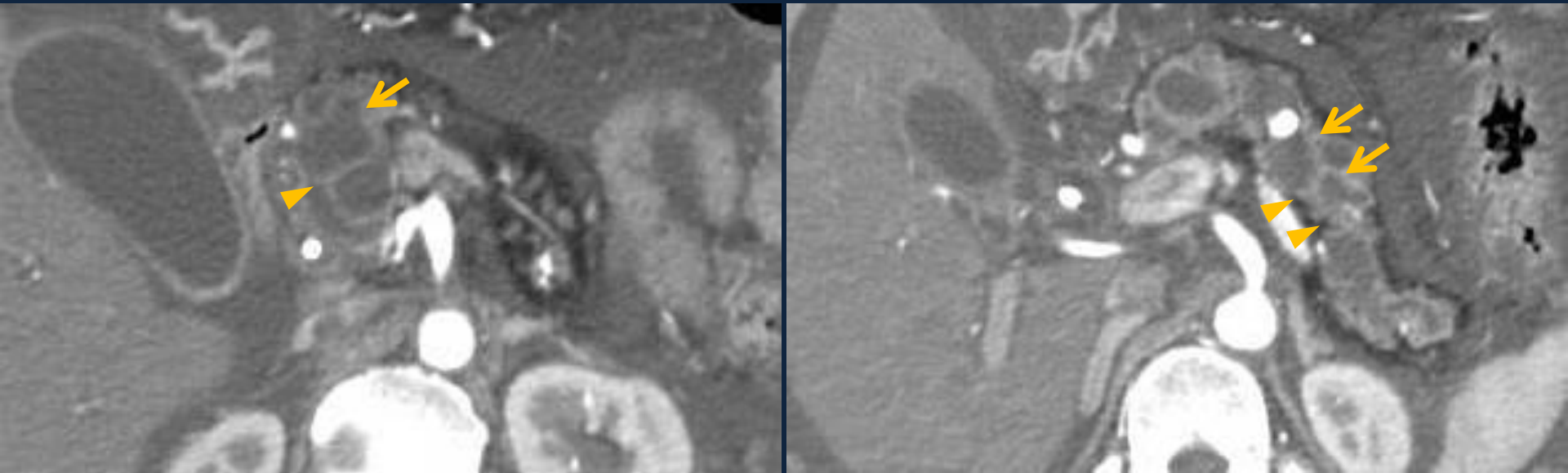
IPMN

- ✓ Mucin producing tumor MD , BD or mixed type
- ✓ M:F = 3:2 (6-7th decade)
- ✓ Location: head >> tail and corpus
- ✓ Communication with MPD
- ✓ The most likely explanation for the calcifications in an IPMN is the presence of mucin, which has a tendency to build up calcium salt deposits. The patients also tend to have underlying chronic calcific pancreatitis
- ✓ Calcifications are reported in 20% of IPMNs. Punctate calcification is the most common pattern (87%), followed by coarse calcification (33%)
- ✓ HPE subtypes: *gastric, intestinal, pancreatobiliary, oncocytic*



81-year-old-male: Axial CECT images in a histopathologically proven case of IPMN reveal diffuse dilatation of MPD (solid arrows) with multiple strictures (arrow heads) and *intraductal calculi* (dotted arrow) mimicking chronic pancreatitis

IPMN

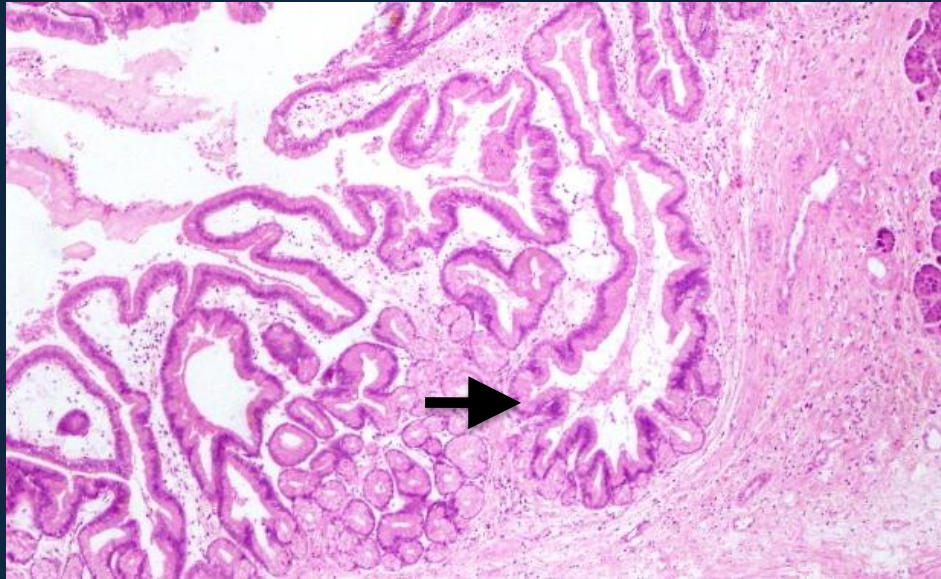


81-year-old-male with IPMN: Axial CECT images reveal diffuse dilatation of MPD (solid arrows) with multiple strictures (arrowheads) and calculi (dotted arrow) mimicking chronic pancreatitis



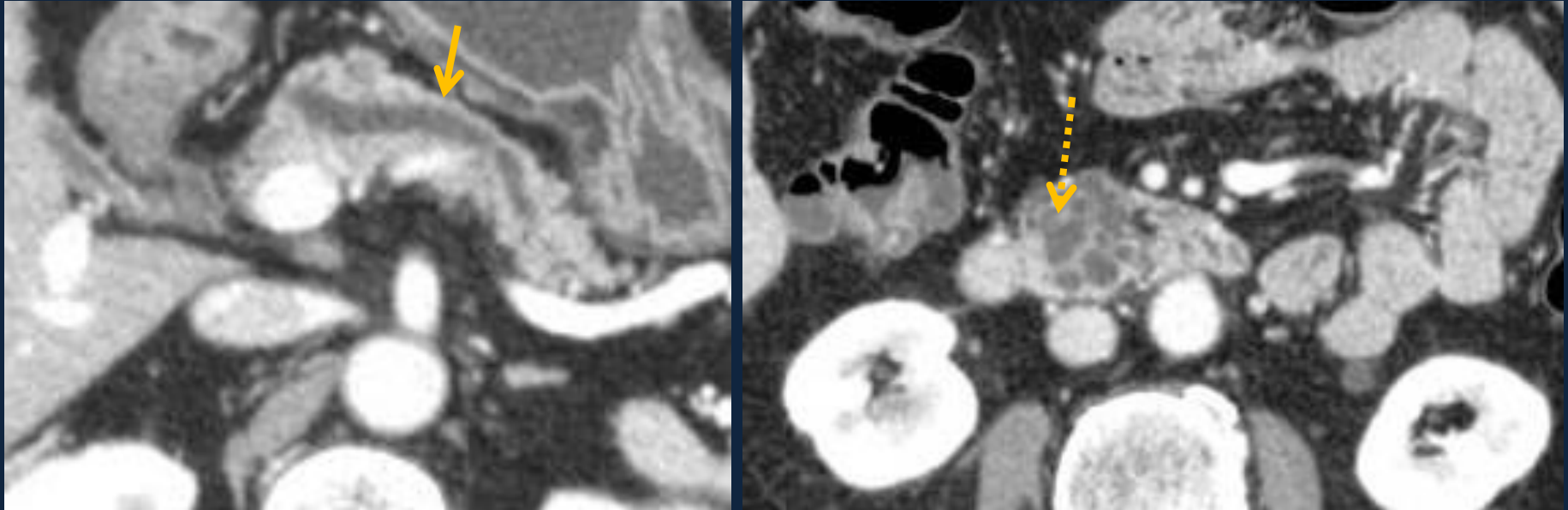
Endoscopy image showing bulging papilla

IPMN



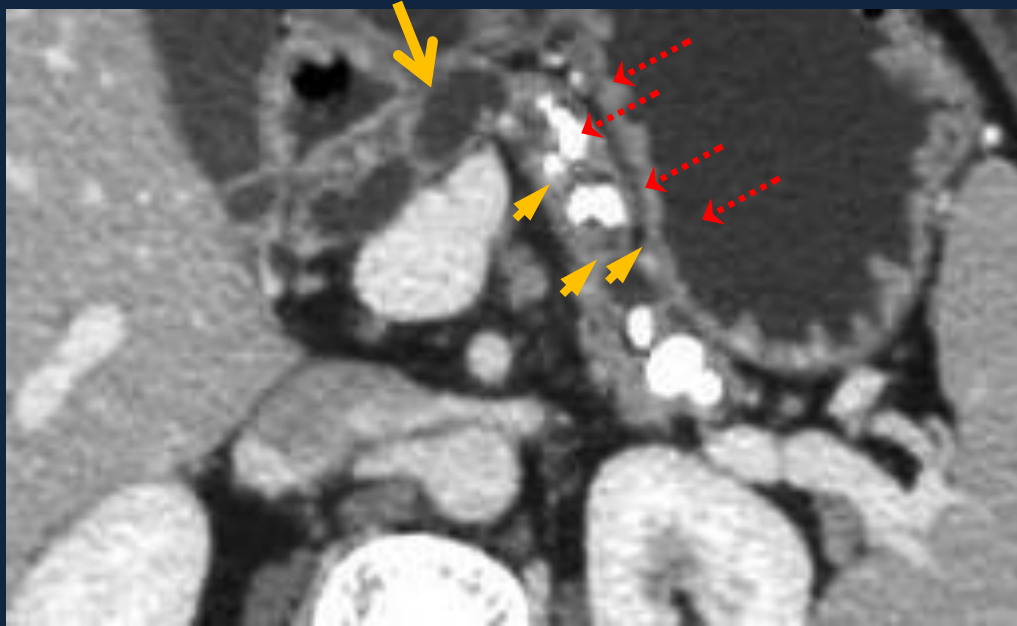
Histopathology sections reveal cystically dilated duct with epithelial infoldings; lined by mucin containing cells and show minimal atypia

IPMN



63-year-old-male with mixed type IPMN: Axial CECT images showing smooth dilatation of MPD in head (solid arrow), body and tail without strictures and grape-like cyst (dotted arrows) in the head and uncinate process of pancreas

Chronic Pancreatitis



36-year-old-male with CP: Axial CECT reveals diffuse dilatation of MPD (solid arrows) with multiple strictures (arrow heads) and calculi (dotted arrow)

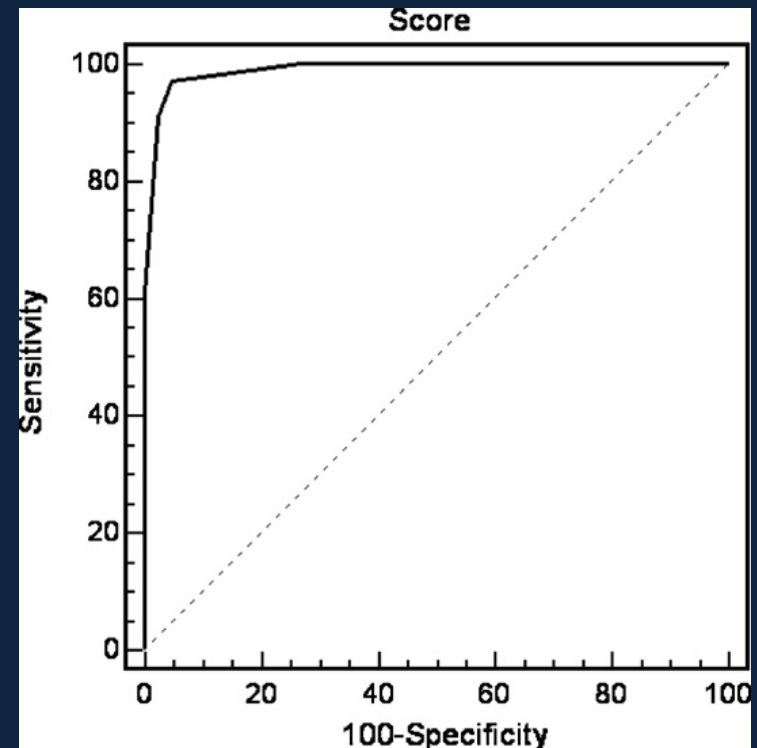
Intraductal papillary mucinous neoplasm of the pancreas: Differentiate from chronic pancreatitis by MR imaging

Jung Hoon Kim^{a,*}, Seong Sook Hong^b, Young Jae Kim^b, Jeong Kon Kim^c, Hyo Won Eun^d

Highly specific findings for IPMN include duct dilatation without stricture, bulging ampulla, nodule in a duct, grape-like cyst shape, and nodule in a cyst.

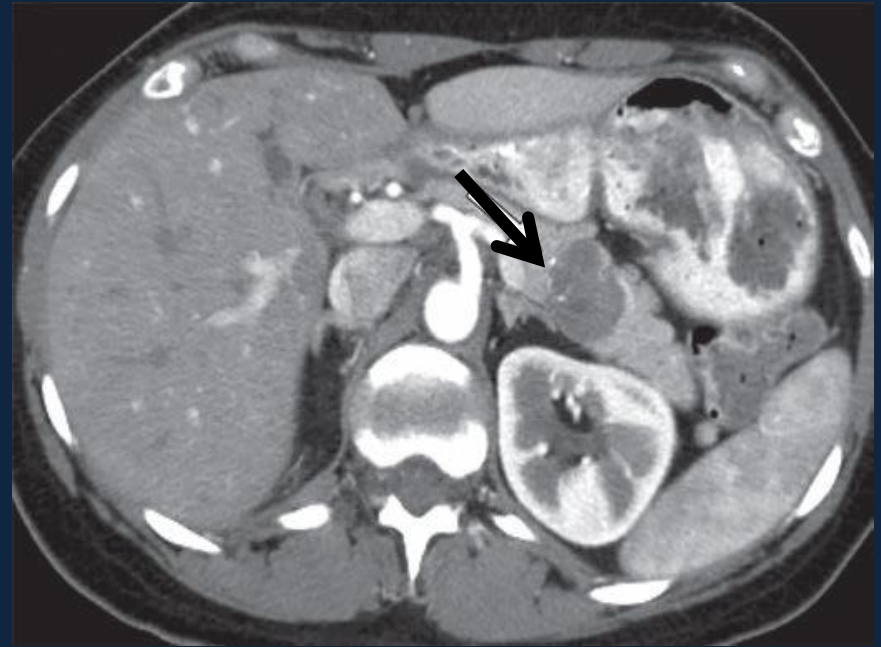
MRI is very useful for differentiating IPMN from chronic pancreatitis using these specific findings.

ROC curve - for the differentiation of IPMN from chronic pancreatitis: The area under the curve is 0.989, respectively.



Solid Pseudopapillary Neoplasm (SPEN)

- ✓ Females – 2-4th decade (Daughter)
- ✓ Solid-cystic neoplasm (Body, tail)
- ✓ Intratumoral hemorrhage
- ✓ Capsule
- ✓ *'Hemangioma-like enhancement'*
- ✓ Calcifications are common, reported in 30% of cases, peripheral and punctate
- ✓ Worrisome features- Lymphadenopathy, invasion
- ✓ In extremely rare instances, SPEN are seen in men, in whom the tumors tend to be larger, have a lobulated contour, and are progressively enhancing



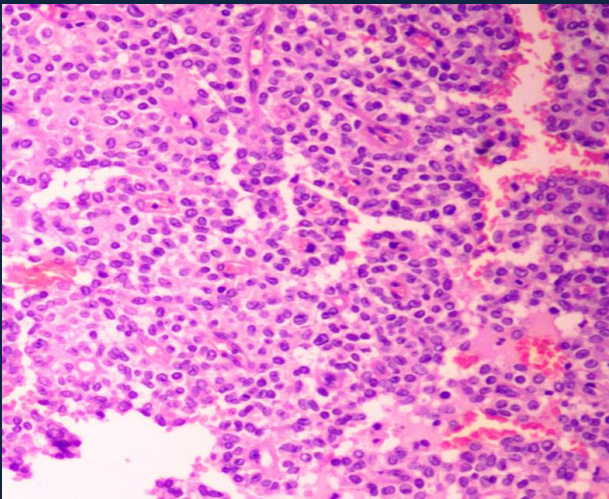
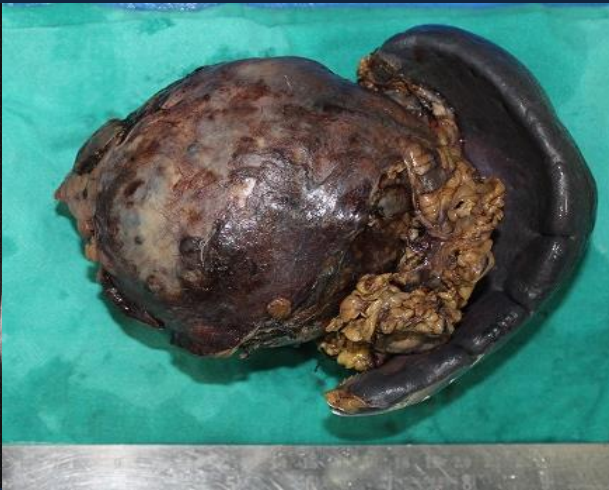
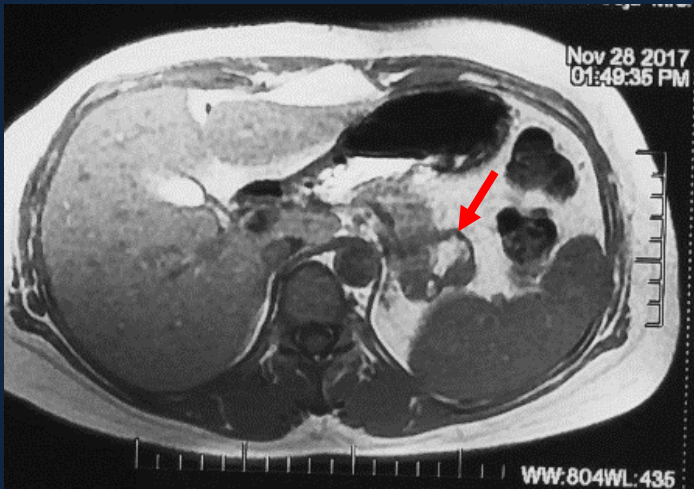
Axial CECT images in a histopathologically proven case of SPEN showing *punctate peripheral calcifications*

Solid Pseudopapillary Neoplasm (SPEN)



34-year-old-female: Axial contrast-enhanced CT images showing iso-attenuating lesion in tail of pancreas (arrows) with peripheral curvilinear calcification.

Solid Pseudopapillary Neoplasm (SPEN)



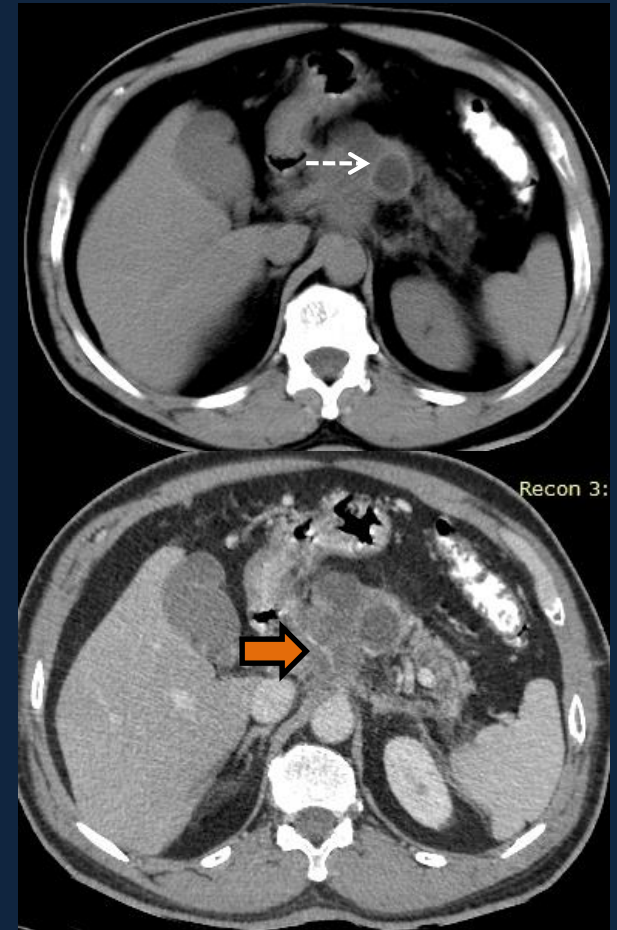
34-year-old-female-same patient as above:

Axial T1-MR image showing hyperintensity within the lesion s/o hemorrhage (arrow) and T2-MR image showing heterogeneous signal with hyperintensity within the lesion.

Post-operative gross specimen. HPE sections showing mainly papillae, round to oval nuclei, fine chromatin, inconspicuous nucleoli, extensive haemorrhage, foamy histiocytes and low mitotic index -revealing features of **SPEN**

Pancreatic adenocarcinoma

- ✓ Pancreatic adenocarcinoma is the most common pancreatic malignancy and typically does not calcify
- ✓ Calcifications in adenocarcinoma can be explained by the occurrence of adenocarcinoma on top of preexisting chronic CP
- ✓ The presence of calcifications can also be caused by pancreatic ductal obstruction by adenocarcinoma
- ✓ Cystic degeneration – 8%
- ✓ Irregular contour, mural nodule, dilatation of MPD, infiltration
- ✓ Lymph nodes, vascular invasion

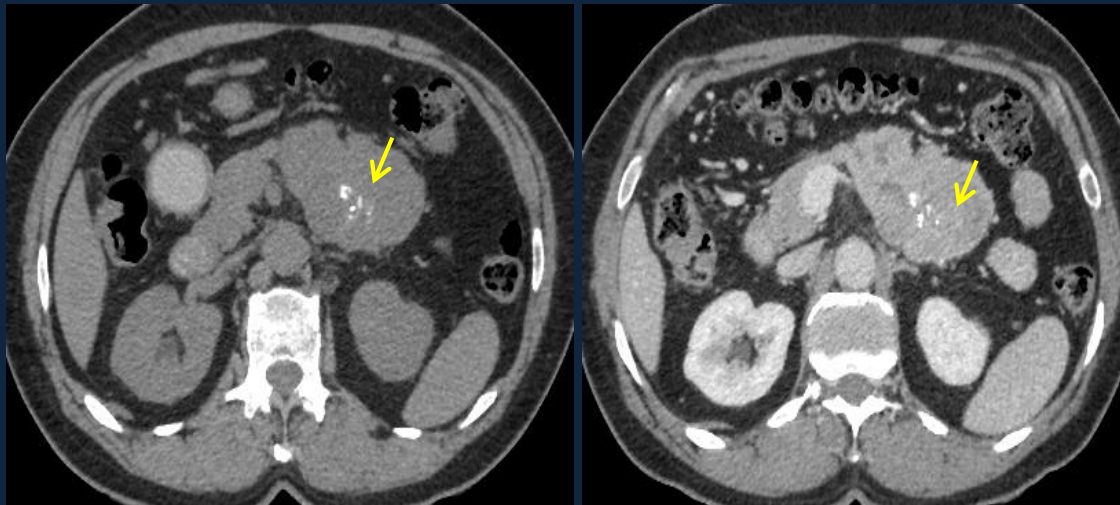


55-year-old-male: Axial CT images showing infiltrating pancreatic ductal adenocarcinoma (arrows) developing in the background of chronic pancreatitis showing subtle *peripheral calcification* (dashed arrows)

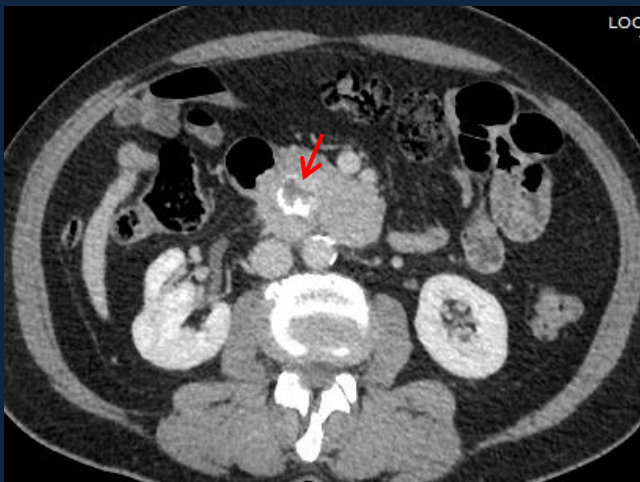
Neuroendocrine tumor

- ✓ NETs - hyperfunctioning and nonhyperfunctioning types on the basis of clinical presentation and hormonal production.
- ✓ Hyperfunctioning tumors tend to be small, frequently smaller than 2 cm in diameter, and present earlier owing to symptoms caused by hormone production.
- ✓ Functioning NETs, such as insulinomas - calcifications in approximately 20%
- ✓ Nonhyperfunctioning NETs, however, tend to present later as larger tumors
- ✓ Nonhyperfunctioning NETs contain calcifications more commonly
- ✓ The calcifications are typically located centrally within the large mass, and the pattern can be coarse, focal, and irregular

Neuroendocrine tumor



51-year-old-male: Axial CT images in a histopathologically proven case of neuroendocrine tumor of pancreas showing *coarse central calcification* (arrow) within the mass in tail of pancreas



Second case: 66-year-old-male: Axial CT images in a histopathologically proven case of neuroendocrine tumor of pancreas showing *coarse central calcification* (arrow) within the mass in head of pancreas

Neuroendocrine tumor

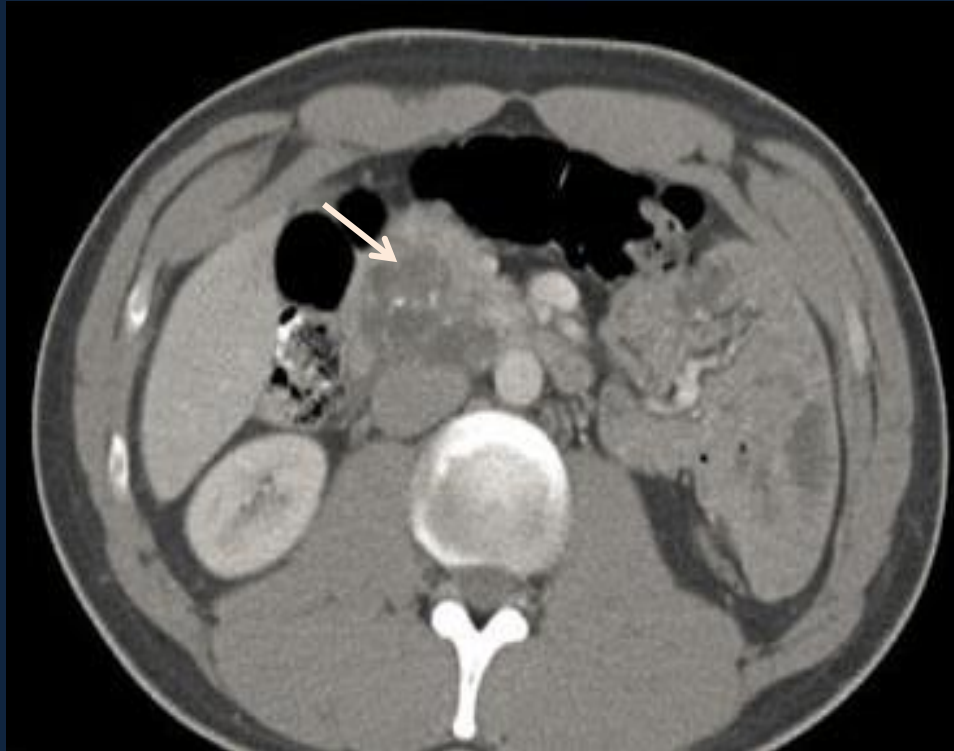


Third case: 58-year-old-female: Axial non-contrast CT images in a histopathologically proven case of neuroendocrine tumor of pancreas showing *fine calcification* (*white arrows*) within the mass in head of pancreas. CECT images showing heterogeneous enhancement with changes of chronic pancreatitis in the form of atrophied pancreatic parenchyma and dilated upstream main pancreatic duct (red arrows). CBD stent is seen in situ (arrowhead)

Acinar cell carcinoma

- ✓ Acinar cell carcinoma is a rare malignant exocrine pancreatic tumor.
- ✓ It typically occurs in men in the 5th-7th decades of life and accounts for less than 2% of primary pancreatic neoplasms.
- ✓ 15% of patients may present with a paraneoplastic lipase hypersecretion syndrome, which results in polyarthralgia, subcutaneous fat necrosis, and peripheral eosinophilia due to the increased secretion of serum lipase.
- ✓ Most of the tumors are exophytic masses (> 5cm) with an enhancing capsule without pancreatic ductal dilatation, which differentiates them from adenocarcinoma.
- ✓ Calcifications have been reported in 6–50% of patients.
- ✓ Acinar cell carcinoma has a more indolent course and better prognosis than pancreatic adenocarcinoma, but it has a higher rate of recurrence.

Acinar cell carcinoma



25-yr-old male: Axial CECT image showing multicystic exophytic lesion in head of pancreas without ductal dilatation and *fine calcifications in the center* mimicking serous cystic neoplasm but on HPE it turned out to be acinar cell cystic neoplasm of pancreas.

Cystic Fibrosis

- ✓ Cystic fibrosis (CF) is an autosomal recessive multisystem disease
- ✓ Viscous epithelial secretions obstruct the proximal pancreatic ducts; functioning acinar cells are replaced with adipose tissue and then with fibrotic tissue.
- ✓ Lipomatous pseudohypertrophy or fatty replacement of the pancreas is the most common imaging finding in CF.
- ✓ Calcifications are less common with an incidence of 8% of cases. Calcifications in the dilated pancreatic duct are caused by a high serum concentration of calcium.
- ✓ Diffuse parenchymal calcifications and pancreatic atrophy - seen in patients with advanced pancreatic insufficiency.
- ✓ Associated with calcifications, small unilocular pancreatic cysts measuring 1–3 mm can also be seen; complete replacement of the pancreas with cysts (pancreatic cystosis) rarely occurs.

Cystic Fibrosis



25-yr-old male case of **cystic fibrosis**: Axial CT image through upper abdomen shows innumerable cysts and cystosis *secondary to cystic fibrosis*. Multiple calcific foci (arrows) are scattered throughout pancreatic parenchyma.

Genetic mutation Pancreatitis

- Autosomal dominant disease involving mutation of the cationic trypsinogen gene
- Less than 1% of cases of recurrent and chronic pancreatitis
- As in tropical pancreatitis, acute attacks usually begin in childhood (5–10 years of age)
- The imaging manifestations of hereditary pancreatitis often resemble those of tropical pancreatitis. Chronic variety is characterized by significant pancreatic atrophy, pancreatic calcifications, and calculi
- Hereditary pancreatitis should be suspected in any patient who has suffered at least two attacks of acute pancreatitis for which there is no explanation (absence of anatomic anomalies, ampullary or MPD strictures, trauma, viral infection, gallstones, alcohol consumption, drug use, or hyperlipidemia).

Genetic mutation Pancreatitis

- The diagnosis may also be considered in patients with unexplained (idiopathic) chronic pancreatitis, patients with a family history of pancreatitis in a first- or second-degree relative, and children with an unexplained episode of pancreatitis that required hospitalization.
- Typically shows “bull’s-eye calcification” with non-calcified central core better appreciated in bone window settings
- Mutations of various genes like PRSS1 and CFTR have been demonstrated.
- Hereditary pancreatitis carries a 50- to 70-fold increased risk of pancreatic cancer.

Genetic mutation Pancreatitis



19-yr-old male case of cystic fibrosis: Axial CECT images showing calcifications in pancreatic parenchyma with non-calcified central core giving *bull's eye configuration* (arrows)

Autoimmune Pancreatitis

- ✓ Pancreatic enlargement (Diffuse or Focal)
- ✓ Sausage shaped pancreas
- ✓ Halo/capsule/envelope
- ✓ Delayed enhancement
- ✓ Dotted enhancement
- ✓ No stranding, vascular invasion
- ✓ Duct penetrating sign (focal variant)/ Duct enhancement sign
- ✓ MR – Diffusion and ADC
- ✓ **Calcifications due to chronic pancreatitis**
- ✓ *However, there may be scenarios in which **dual pathology** - AIP and ductal adenocarcinoma coexist in a single patient*



41-yr-old male case of autoimmune pancreatitis on steroids showing small **punctate calcifications** in the pancreatic parenchyma as a sequelae to repeated attacks of chronic pancreatitis and small foci of intrapancreatic necrosis in tail of pancreas

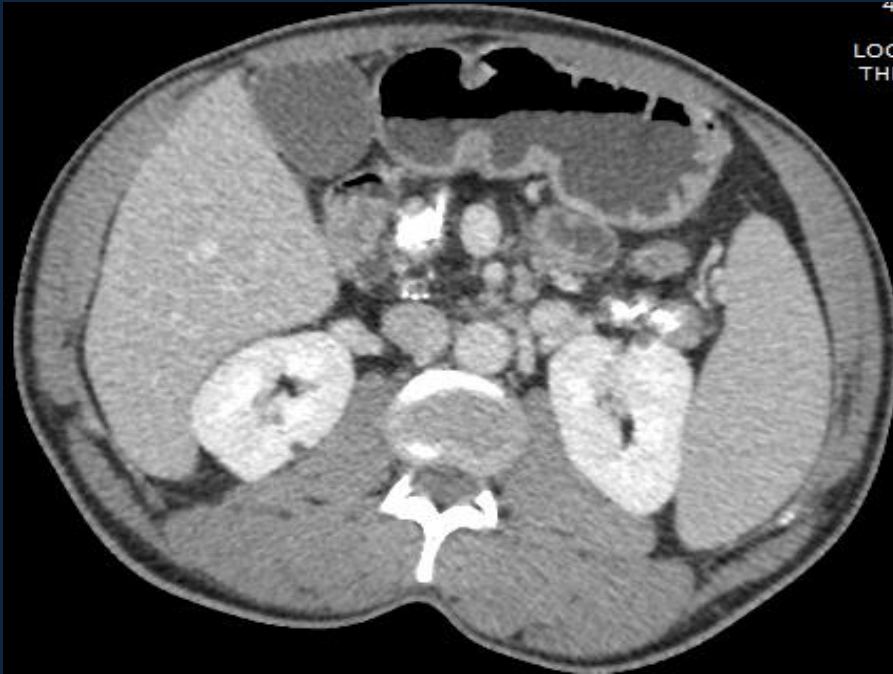
Tropical Pancreatitis

- Young age at onset, association with malnutrition, regional predisposition in tropical countries
- First described in 1959 in Indonesia. Relatively large number of cases occur in the southern state of Kerala in India
- Etiopathogenesis of tropical pancreatitis - PEM, pancreatic ductal anomalies, food toxicities such as chronic cyanide toxicity from cassava, and possible genetic predisposition (SPINK 1 N34S mutation and CFTR mutation)
- Nearly two-thirds of affected patients develop fibrocalculous pancreatic diabetes within a decade of onset

Tropical Pancreatitis

- The characteristic features are absence of association with alcohol consumption, biliary tract disease, and absence of other biochemical or structural predisposing factors for pancreatitis.
- On imaging, multiple large pancreatic calculi within a dilated pancreatic duct is seen. Ductal dilatation and calculi are known to occur in more than 80% of patients, with parenchymal atrophy occurring in nearly 50%. The intraductal calculi in tropical pancreatitis are discrete, dense, and up to 5 cm in size. In contrast, the intraductal calculi in alcohol-related chronic pancreatitis are usually small and speckled.
- Management consists largely of supportive treatment for associated pain, diabetes, and steatorrhea.

Tropical Pancreatitis



36-yr-old male, non - alcoholic: Axial CECT images showing shows a markedly dilated and tortuous MPD with large intraductal stones in a case of *tropical pancreatitis*.

Various Types and unusual causes of Pancreatitis

Types of pancreatitis	Clinical Features	Imaging Features
Autoimmune pancreatitis	Absence of the classic acute attacks of pancreatitis, elevated immune markers, dramatic response to steroid therapy	“Sausage” shape pancreas, peripheral “rind” of hypoattenuation
Groove pancreatitis	Duodenal and biliary obstruction; symptoms overlap with those of pancreatic cancer, frequently leading to misdiagnosis	Soft tissue within the pancreaticoduodenal groove , small cysts along medial duodenal wall
Tropical pancreatitis	Young age, associated with malnutrition, regional predisposition in tropical countries, increased risk of adenocarcinoma	Multiple large discrete calculi up to 5 cm within a dilated pancreatic duct (>80% of cases)

Various Types and unusual causes of Pancreatitis

Types of pancreatitis	Clinical Features	Imaging Features
Hereditary pancreatitis	Young age, at least 2 attacks of pancreatitis with no underlying cause, family history of pancreatitis in a first- or second-degree relative	Acute: nonspecific; chronic: significant pancreatic atrophy, pancreatic calcifications, and calculi
Pancreatitis in ectopic/ Heterotopic pancreas	Pancreatitis rarely diagnosed clinically and radiologically; may manifest as abdominal pain (77%), abdominal fullness (30%), or malena (24%)	CT: oval or round masses in the gastric wall that follow the enhancement pattern of pancreatic tissue
Pancreatitis in cystic fibrosis	Exocrine pancreatic insufficiency, full-blown acute pancreatitis rare (1.2%), pancreatitis may be the first manifestation of cystic fibrosis	Fatty replacement of pancreas, calcification (7%), and cyst formation (pancreatic cystosis)

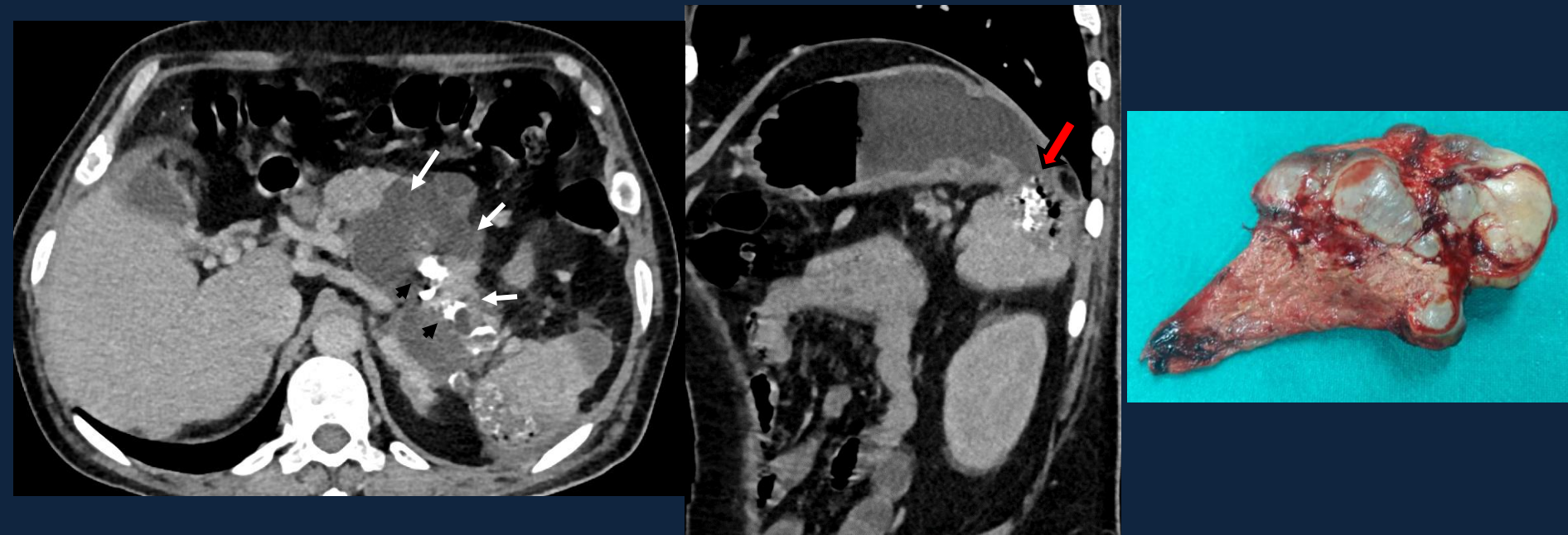
Calcified calculi

- ✓ Calcified calculi can be seen within biliary and pancreatic ducts.
- ✓ These appear as high-attenuation foci on contrast-enhanced CT images and can be misinterpreted as enhancing masses.
- ✓ Thus, reviewing unenhanced CT images is important to confirm that these are calcified calculi rather than enhancing lesions.



31-yr-old male case of chronic calcific pancreatitis showing *dense coarse intraductal calculi* within the dilated pancreatic duct

Gastro-splenic fistula resulting into Splenic abscess



47-year-old-male with past history of glue embolization of splenic artery aneurysms presenting with fever, vomiting and pain abdomen: Axial CECT image showing changes of necrotizing pancreatitis (white arrows) with hyperdense glue material (arrowheads) in pancreatic bed. Sagittal reformatted image showing gastro-splenic fistula (red arrow) and hypoattenuating collection with glue particles in the splenic parenchyma. Post-operative specimen of the patient.

“CYSTIC” Model approach in cystic pancreatic lesions

Pancreatic cystic lesions	‘C’	‘Y’	‘S’	‘T’	‘I’	‘C’
	Characteristic location and loculation	Years-age group	Sex Size Shape	Tumour enhancement pattern	Incidence Imaging prognosticators for malignancy	Communication with duct Calcification Central scar
Pseudocyst	Head, body, tail Unilocular/multilocular	Any age group	M>F Any size Variable shape	No enhancement Enhancing wall s/o infection	Very common No malignant potential	Disconnected duct syndrome Rim calcification ± No scar
MCN	Body, tail Macrocytic (< 6, each > 2 cm)	4 th -5 th decade	F>>M Any size Oval	Thick peripheral wall enhancement, subtle thin septal enhancement	Moderately common Solid enhancing component; irregular wall; peripheral calcification	Absent Peripheral calcification No scar
BD-IPMN	Head, body, tail Macrocytic	6 th -7 th decade	M>F (3:2) Usually < 3cm Bunch of grapes	Thin peripheral enhancement ±	Common Size > 3 cm; MPD > > 6 mm; thick irregular wall; septa; mural nodules	Present Septal calcification ± No scar
MD-IPMN	Head, body, tail Diffuse MPD dilatation	6 th -7 th decade	M>F (3:2) MPD ≥ 5mm Elongated	No enhancement	Rare MPD > 10 mm; mural enhancing nodules	Present Calcification ± No scar
SCN	Head, body, tail Microcystic (> 6, each < 2 cm)	6 th -7 th decade	F > M (3:1) Variable Lobulated	Thin wall and septal enhancement	Moderately common Rarely malignant - enhancing solid component; nodes	Absent Central calcification Central scar present

"CYSTIC" Model approach in cystic pancreatic lesions

Pancreatic cystic lesions	'C'	'Y'	'S'	'T'	'I'	'C'
	Characteristic location and loculation	Years-age group	Sex Size Shape	Tumour enhancement pattern	Incidence Imaging prognosticators for malignancy	Communication with duct Calcification Central scar
SPN	Body, tail Unilocular	2 nd -4 th decade	F>>>M Variable Oval/Round	Hemangioma like progressive enhancement	Rare Large size; local Invasion; enlarged nodes	Absent Uncommon No scar
Cystic NET	Head, body, tail	5 th -6 th decade	F=M Variable Oval	Thick peripheral; solid-cystic enhancement	Moderately common Large size; invasion; metastasis	Absent Calcification ± Absent
Lympho-epithelial cysts	Extrapancreatic/exophytic Variable shape	3 rd -8 th decade	M>>F (4:1) Variable Lobulated	Negative or low CT HU value Hyperintense T1-W MR Subtle diffusion restriction	Very rare No malignant potential	Absent Septal/peripheral calcification ± Absent
Lymphangioma	Body, tail Macrocystic	Any age group	F>M Usually large Variable	Predominantly cystic with very thin septations	Very rare No malignant potential	Absent Septal calcification ± Absent
Cystic pancreatic hamartoma	Head, body, tail Unilocular/multilocular	3 rd -6 th decade	M>F Variable Variable	Solid-cystic, chronological change in morphology	Extremely rare No malignant potential	Absent Variable Absent

“CYSTIC” Model approach in cystic pancreatic lesions

Pancreatic cystic lesions	‘C’	‘Y’	‘S’	‘T’	‘I’	‘C’
	Characteristic location and loculation	Years-age group	Sex Size Shape	Tumour enhancement pattern	Incidence Imaging prognosticators for malignancy	Communication with duct Calcification Central scar
Dermoid cyst	Head, body, tail Unilocular/multilocular	Any age group	M=F Variable Variable	Fat, calcium, Negative CT HU	Extremely rare No malignant potential	Absent Usually present Absent
Epidermoid cyst	Tail Unilocular	4 th decade	M=F Small Smooth	Enhancement similar to splenic parenchyma; accessory spleen	Extremely rare No malignant potential	Absent Absent Absent
Retention cyst	Head, body, tail Unilocular	3 rd -7 th decade	M=F Variable Smooth	Variable peripheral enhancement; ductal anomaly; stricture/stone/tumour	Extremely rare Solid component, macro-septations, mural nodules	Present – uniform upstream dilatation Variable Absent
Dysontogenetic cyst	Diffuse involvement Solitary or polycystic	Congenital	M=F Variable Variable	Simple cysts without nodule/septa/wall thickening/enhancement	Extremely rare No malignant potential	Absent Absent Absent
Duplication cyst	Head, body, tail Unilocular	Any age group	M=F Small-medium size Smooth	Cyst with smooth enhancing walls; Associated with anomalies of pancreas and ducts	Rare No malignant potential	Communication ± Absent Absent

Fluid Analysis

Fluid Characteristics	Pseudocyst	SCN	MCN	IPMN	SPN	CPEN
Cytology	No epithelium	Cuboidal epithelium	Columnar epithelium	Columnar epithelium	Branching papilla, myxoid stroma	Salt-and-pepper chromatin
Viscosity	Low	Low	High	High	Bloody fluid	Low
Mucin	Low	Low	Very High	Very High	NA	Low
CEA	Low	Low	High	High	NA	Low
CA 19-9	Low	Low	High	Variable	NA	Unknown
CA 72-4	Low	Low	High	High	NA	Unknown
Amylase	Very high	Low	Variable	High	Low	Low
Glycogen	Absent	Very High	Absent	Absent	NA	Absent
DNA <u>KRAS</u>	Absent	Absent	Present	Present	Unknown	Unknown

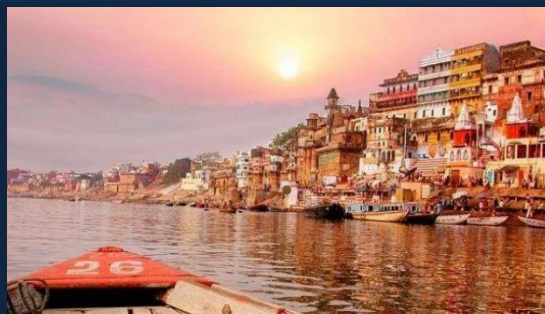
NA: Not Applicable; SCN: Serous cystic neoplasm; MCN: Mucinous cystic neoplasm; IPMN: Intraductal-papillary mucinous neoplasm; SPN: solid pseudopapillary neoplasm; CPEN: cystic pancreatic endocrine neoplasm

Conclusion

- ❖ A wide spectrum of pathologic conditions affecting the pancreas can cause cystic/necrosis with calcifications.
- ❖ A pattern recognition approach entailing the morphologic characteristics of the pancreatic lesion and pattern of cystic changes/necrosis with calcification can be helpful in making a reasonable differential diagnosis and reaching a specific diagnosis, because the management of these lesions varies.
- ❖ Imaging studies should be interpreted in the clinical context in conjunction with relevant laboratory findings.

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