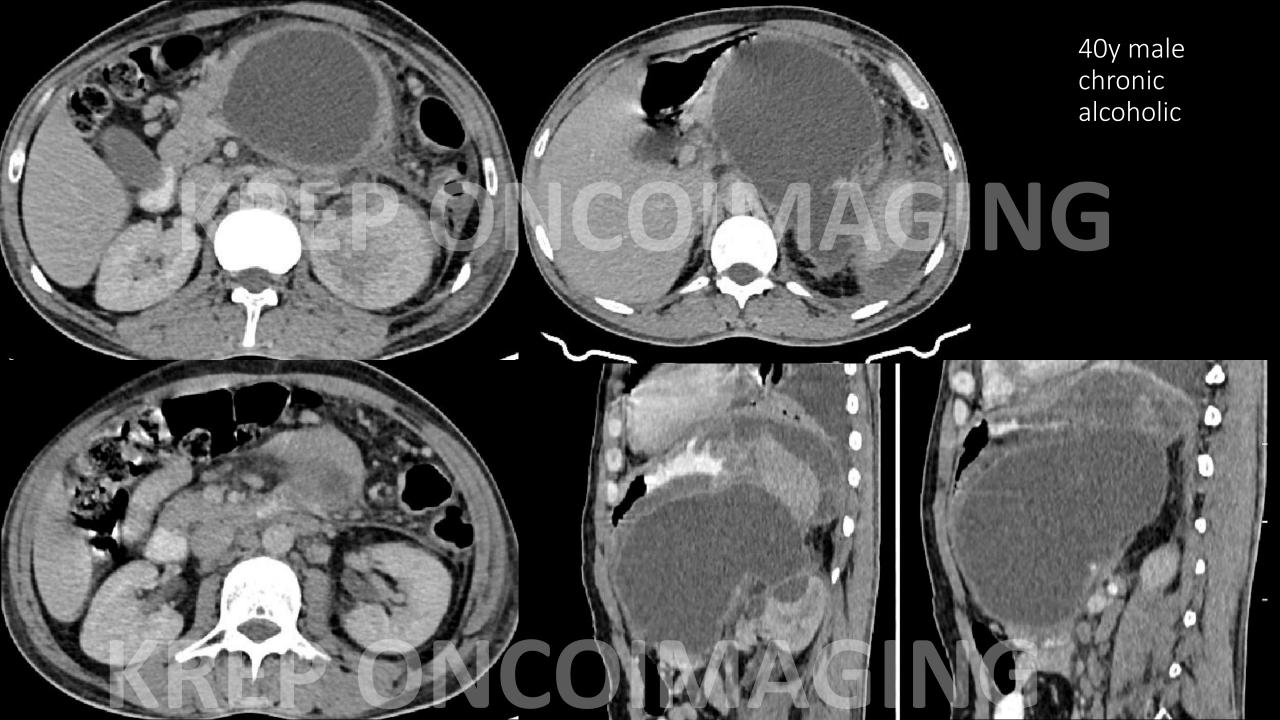


2025

KARNATAKA RADIOLOGY EDUCATION PROGRAM



- Large rim enhancing fluid attenuating area in region of body and tail of pancreas measuring about 10 x 12 x 15 cm. Wall thickness measures 0.25 mm. No mural nodule. No internal enhancing areas. No isodense or hyperdense contents. No gas foci.
 - A segment of splenic artery is seen within the lesion.
 - Anteriorly there is mass effect upon stomach (wall shows hypoenhancing mural thickening possibly submucosal edema / inflammatory etiology)
 - Posteriorly there is extension into left paranephric space with a small component compressing left renal upper pole parenchyma.
- Moderate left pleural effusion is noted. Minimal ascites was noted.
- The visualized pancreatic parenchyma shows normal enhancement. MPD was not dilated. No calcific foci within.
- A large perisplenic collection is noted. Spleen otherwise is normal in size and shows normal enhancement.
- Adjacent omental fat stranding was noted. No nodularity.
- Multiple prominent and few enlarged para-aortic lymph nodes are noted showing homogeneous enhancement, likely reactive.

- There are no features to suggest cystic neoplasm, this likely represents a pancreatic pseudocyst with perisplenic and paranephric collection, moderate left pleural effusion, ascites; mCTSI 6/10.
- Gastric mural hypoenhancing thickening is mostly submucosal edema, can be correlated clinically and followed up with a USG before discharge to document resolution.
- This person is prone for pseudoaneurysm development.

1. Definition & Pathophysiology:

- A well-circumscribed, encapsulated fluid collection rich in pancreatic enzymes, arising as a delayed (>4 weeks) complication of acute or chronic pancreatitis.
- Lacks an epithelial lining (hence pseudocyst); walls composed of granulation and fibrous tissue.

2. Etiology:

- Most commonly associated with alcoholic pancreatitis, gallstone pancreatitis, trauma, or postnecrotic pancreatic duct disruption.
- Chronic pancreatitis produces multiple, recurrent pseudocysts from ductal outflow obstruction.

3. CT Imaging Features (Primary Modality):

- Round or ovoid, well-defined fluid-attenuation lesion with thin or moderately thick enhancing wall.
- May exert mass effect on adjacent organs (stomach, spleen, kidney).
- No internal solid enhancing components (if present → consider walled-off necrosis or cystic neoplasm).
- May contain debris, hemorrhage, or infection causing heterogeneous attenuation.

4. MRI Characteristics:

- T1: Low signal unless hemorrhagic/high protein content.
- · T2: Homogeneously hyperintense fluid cavity.
- Post-contrast: Thin peripheral rim enhancement.
- MRCP helps identify pancreatic duct communication, strictures, and ductal leaks.

5. Distinguishing from Other Pancreatic Collections:

- Acute peripancreatic fluid collection (<4 weeks): no wall.
- Walled-off necrosis (WON): thick wall + non-enhancing solid necrotic debris.
- Cystic neoplasm (MCN/SCN/IPMN): internal septa, nodules, calcification, or enhancement.
- Abscess: gas, thick irregular wall, clinical sepsis.

6. Complications (Key Imaging Roles):

- Infection (air, thick irregular wall).
- Hemorrhage (high T1 signal, pseudoaneurysm).
- Rupture into peritoneal cavity or GI tract.
- Biliary or gastric outlet obstruction from mass effect.
- Vascular compression or thrombosis (splenic vein, portal vein).

7. Management Implications:

- · Asymptomatic pseudocysts are often observed.
- Intervention indicated for symptoms, infection, hemorrhage, persistent >6 weeks, >6 cm, or obstruction.
- Endoscopic ultrasound-guided drainage preferred; CT/MR defines wall maturity and vascular proximity (to avoid pseudoaneurysm bleed).

8. Oncoradiologic Reporting Essentials:

- Size, location, wall thickness, internal debris, pancreatic duct communication, relation to vessels, signs of necrosis or infection.
- Presence of additional pseudocysts, walled-off necrosis, or pancreatic atrophy/calcifications suggesting chronic pancreatitis.
- Any solid nodules or mural enhancement must be highlighted for exclusion of cystic neoplasm.

Contributors

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