Chronic Pancreatitis

Chronic pancreatitis represents a continuous, prolonged, inflammatory and fibrosing process of the pancreas with *irreversible* morphologic changes resulting in permanent endocrine and exocrine pancreatic dysfunction.

Acute pancreatitis and chronic pancreatitis are assumed to be *different disease processes*, and most cases of acute pancreatitis do not result in chronic disease.

Chronic pancreatitis

- LO1:Pathophysiology
- LO2: Etiology
- LO3: Clinical features
- LO4: Diagnosis
- LO5: Treatment
- LO6: Complications

LO1: PATHOPHYSIOLOGY

Incompletely understood

Why 10% heavy alcoholics develop chronic pancreatitis and the rest not, or limited to asymptomatic pancreatic fibrosis

LO1: Ductal obstruction hypothesis

Pancreatic ductal stone are seen in alcoholic, tropical, hereditary, idiopathic

Sleisenger and Fordtran's - 9th edition.

LO1: Toxic metabolic hypothesis

(alcohol) Direct injurious effect on acinar and ductal cells

LO1: Necrosis fibrosis hypothesis

Repeated episodes of acute pancreatitis with cellular necrosis or apoptosis, healing replaces necrotic tissue with fibrosis

LO2: Causes

- **Toxic-metabolic**

a) **Alcoholic**

b) **Tobacco smoking**

c) **Hypercalcaemia**

d) **Hyperparathyroidism**

e) **Hyperlipidemia (rare and controversial)**

f) **Chronic renal failure**

- **Obstructive**
- **Medications (Phenacetin abuse)**
- **Idiopathic**
- **Genetic**
- **Autoimmune**

LO3: Clinical features

- Chronic pancreatitis is a relapsing condition that presents with abdominal pain, occurring in 95% of cases.
- Pain can be episodic, lasting hours to days, or it can persist for months or even years. The pain is characteristically steady in the epigastrium, and it frequently radiates to the back.
- Weight loss, Steatorrhea.

LO4: Diagnosis

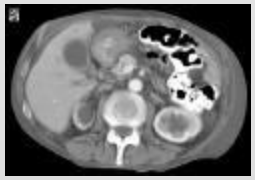
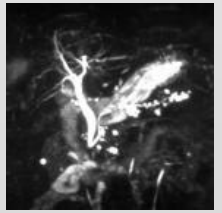
- No single test is adequate
- Tests for function
- Tests for structure
- Both are more accurate in advanced disease .

LO4:Tests of function – hormone stimulation

- Direct Tests
  - Secretin/ secretin CCK test abnormal secretin stimulations test when >60 % affected
- Indirect Tests
  - Fecal elastase< 100mcg/mg stool - severe exocrine insufficiency.
- Fecal chymotrypsin
- Serum trypsinogen (trypsin)< 20ng/ml,
- Blood glucose

Tests of structure

- Plain film of the abdomen
- CT
- Ultrasonography
- MRI, particularly MRCP
- ERCP

LO4: Routine laboratory tests

Serum amylase and lipase

- May be elevated in acute exacerbations
- Also found increased in pseudocyst, ductal stricture, internal pancreatic fistula, pancreatic carcinoma, cholecystitis, ectopic pregnancy

LO4: Classics of Chronic pancreatitis

Pancreatic calcification

Steatorrhea

Diabetes mellitus

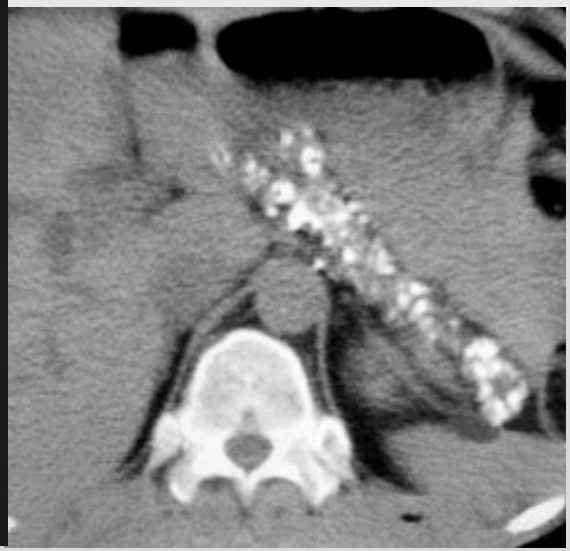
Found in less than a third of pts with CP

- Fecal elastase

LO4: Plain films

- ***Pancreatic calcifications*** **are shown in 25-59% of patients.**
- **This feature is pathognomonic for chronic pancreatitis.**
- **Calcification is punctate or coarse, and it may have a focal, segmental, or diffuse distribution.**

chronic pancreatitis with marked calcification of the pancreatic parenchyma.



LO4: Upper GI tract barium series

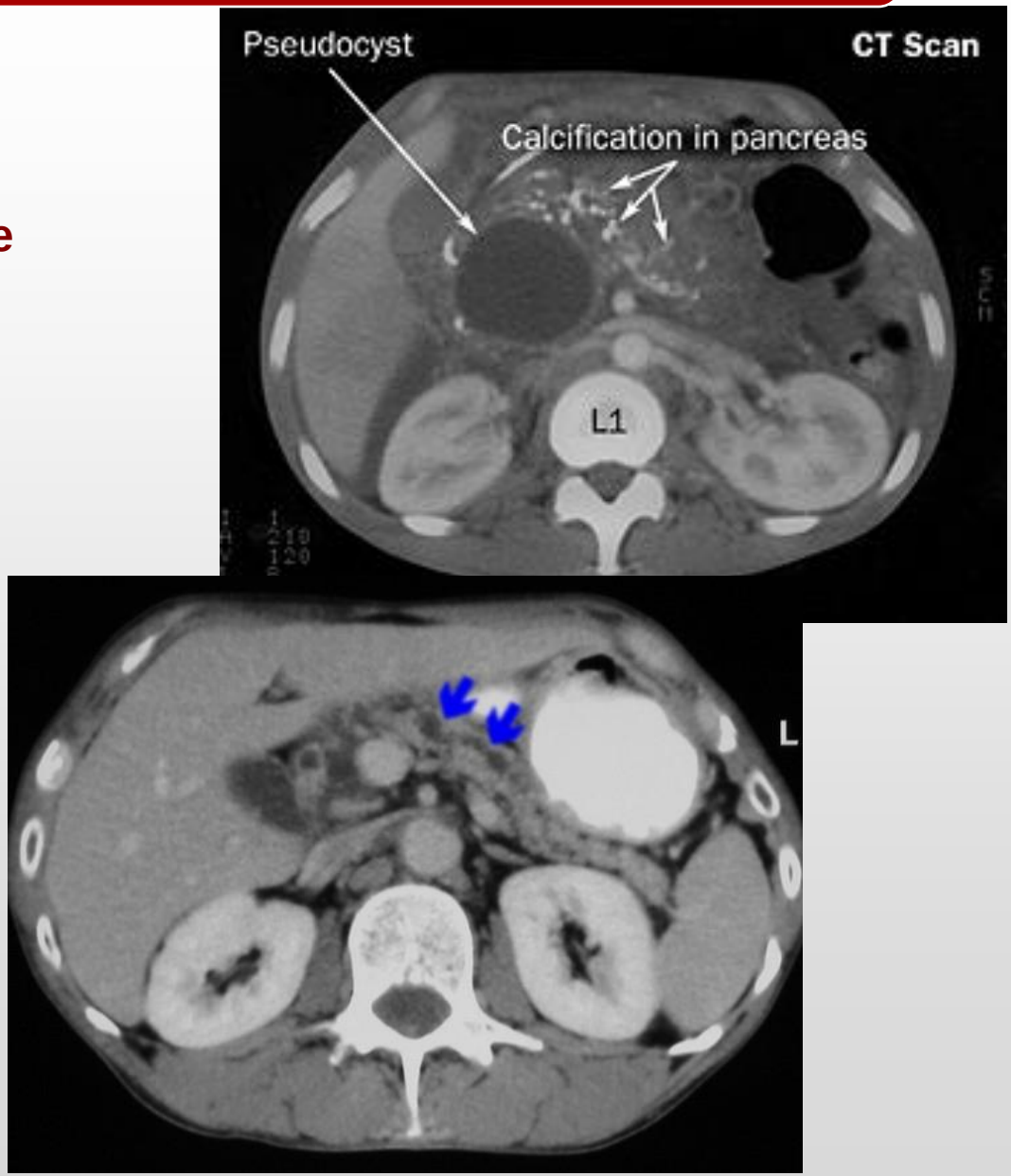
The anatomic proximity of the pancreatic head and stomach antrum is constant, and enlargement of the pancreatic head usually causes effacement of the antrum. This finding has been termed the *pad sign*.

Upper gastrointestinal tract barium study shows a **reverse *3*** in the duodenum due to chronic pancreatitis.

Pancreatic carcinoma can have a similar appearance

LO4: CT Findings

Currently, CT is regarded as the *imaging modality of choice* for the initial evaluation of suggested chronic pancreatitis.



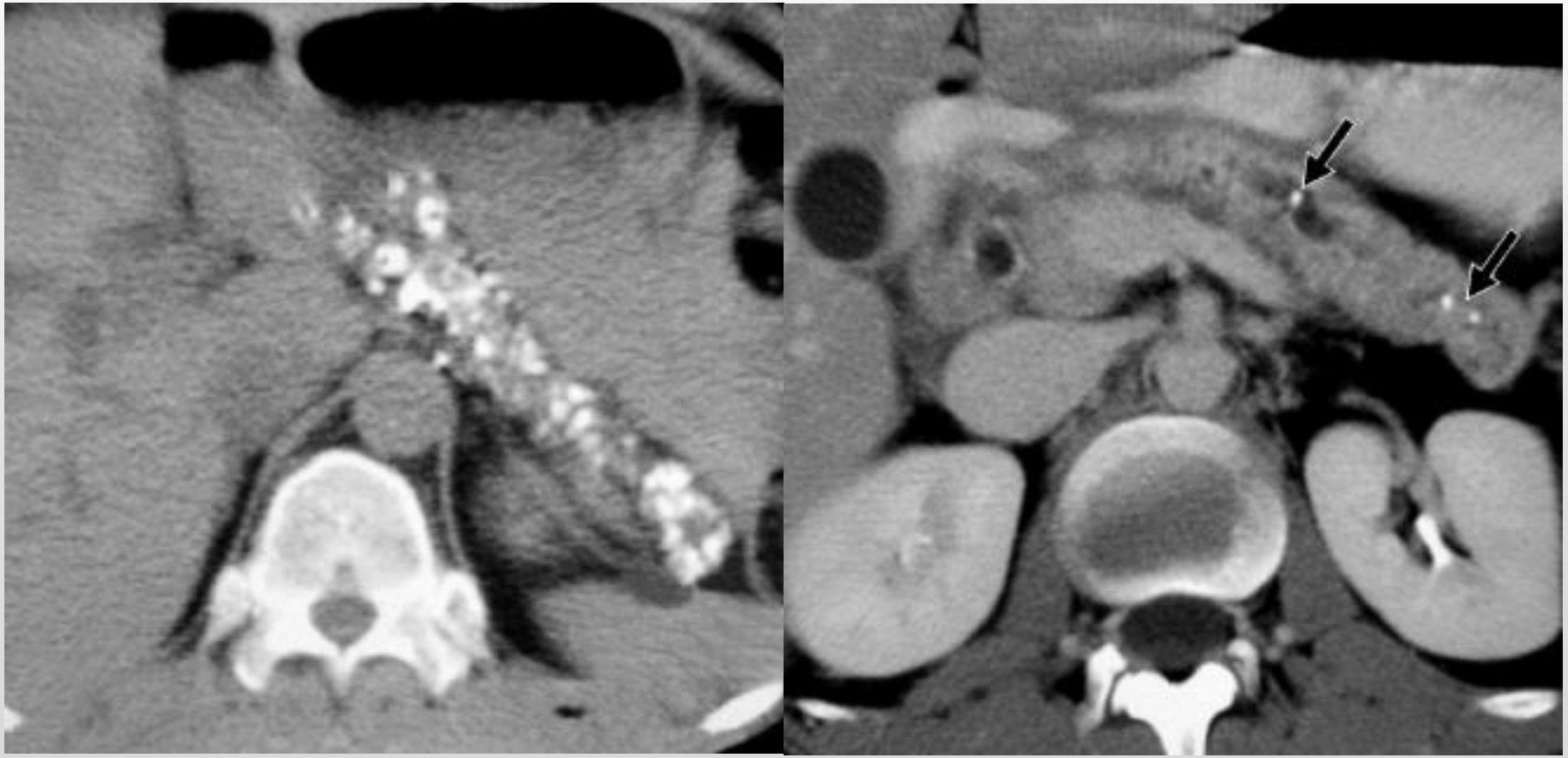
The diagnostic features of:

- **pancreatic enlargement,**
- **pancreatic calcifications,**
- **pancreatic ductal dilatation,**
- **thickening of the peripancreatic fascia, and**
- **bile duct involvement**

are depicted well on CT scans.

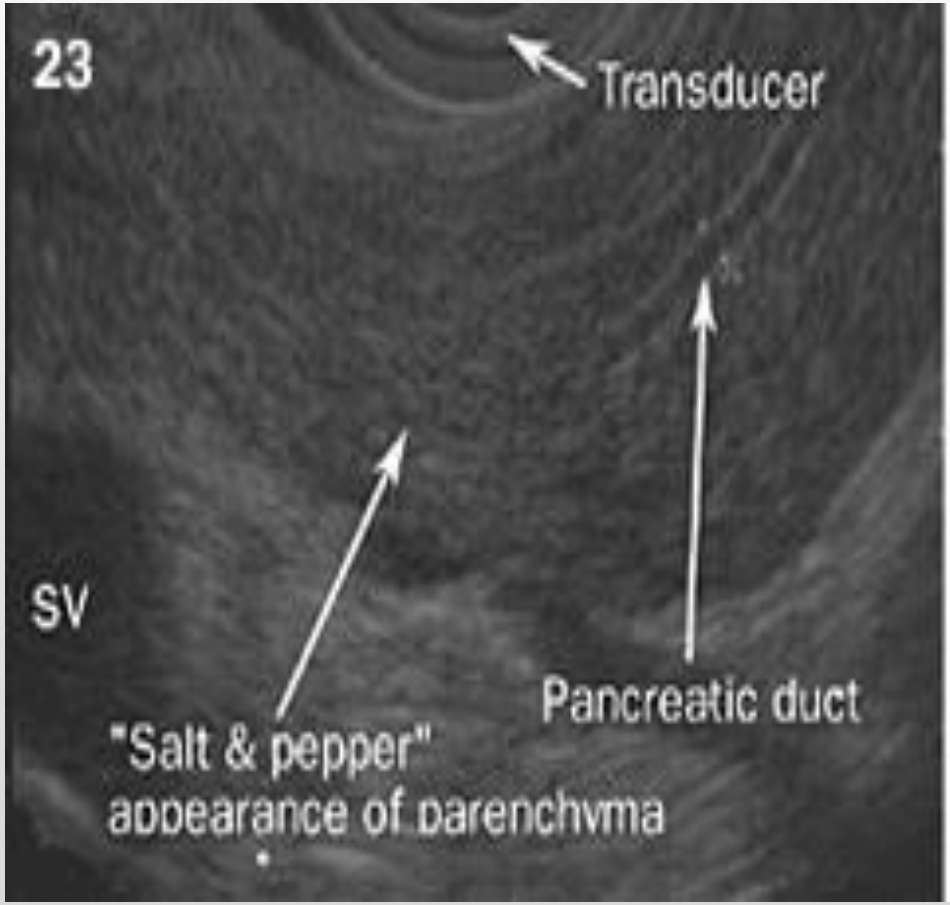
LO4: CT Findings

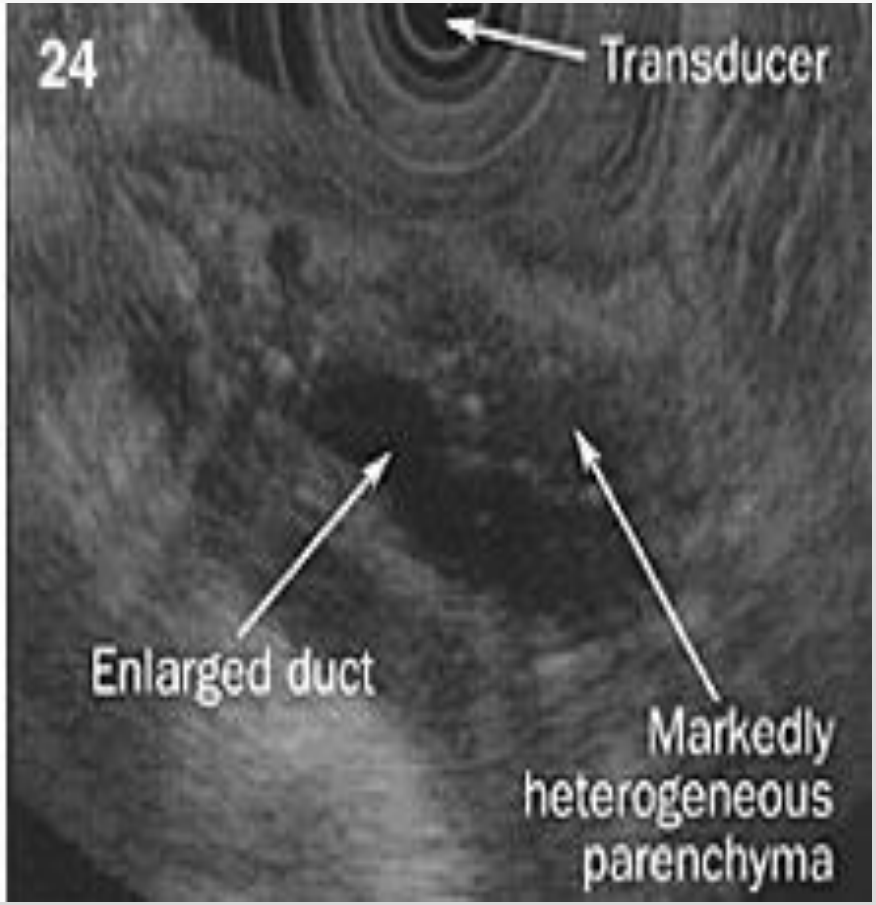
The sensitivity of plain film for detection of *pancreatic calcifications* is about 80 %, which is higher than that of sonography but lower than that of CT.



LO4: ULTRASOUND

- In late stages of the disease, the pancreas becomes atrophic and fibrotic, and it shrinks. These changes result in a small.
- Pseudocyst may occur as masses may mimic pancreatic neoplasia.

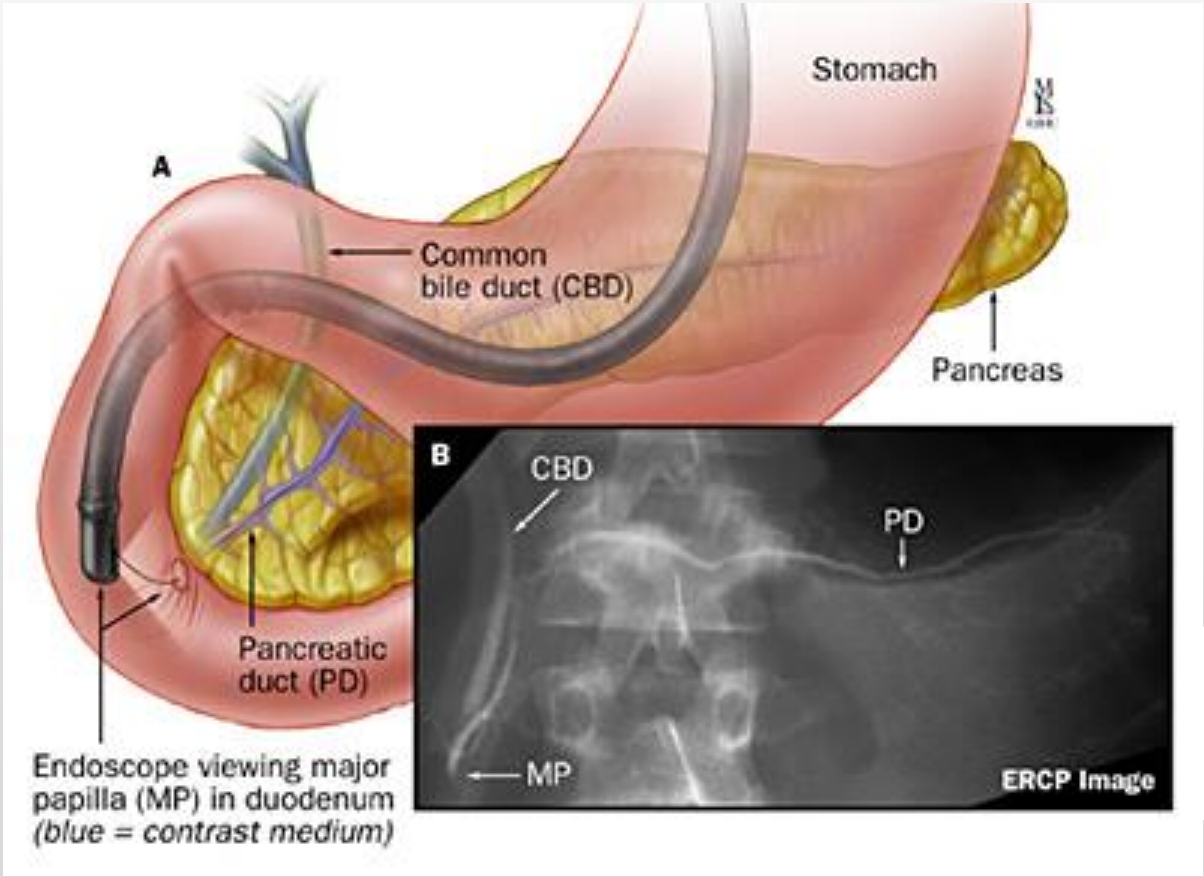




LO4: ERCP

Endoscopic retrograde Cholangiopancreatography (ERCP)

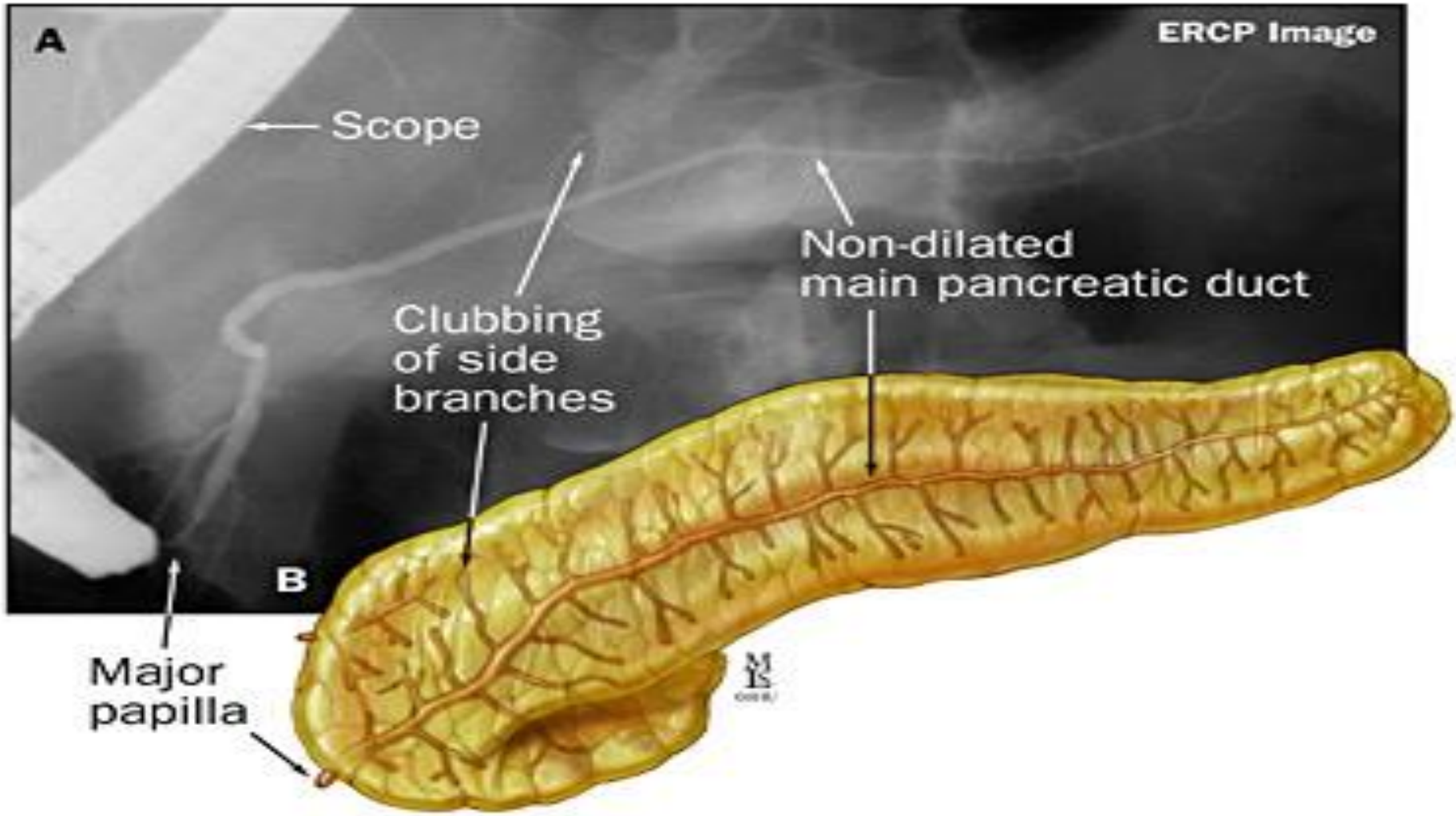
ERCP is the most sensitive and specific technique in the investigation of chronic pancreatitis, although *it is invasive and may cause an acute episode of pancreatitis and ascending cholangitis.*



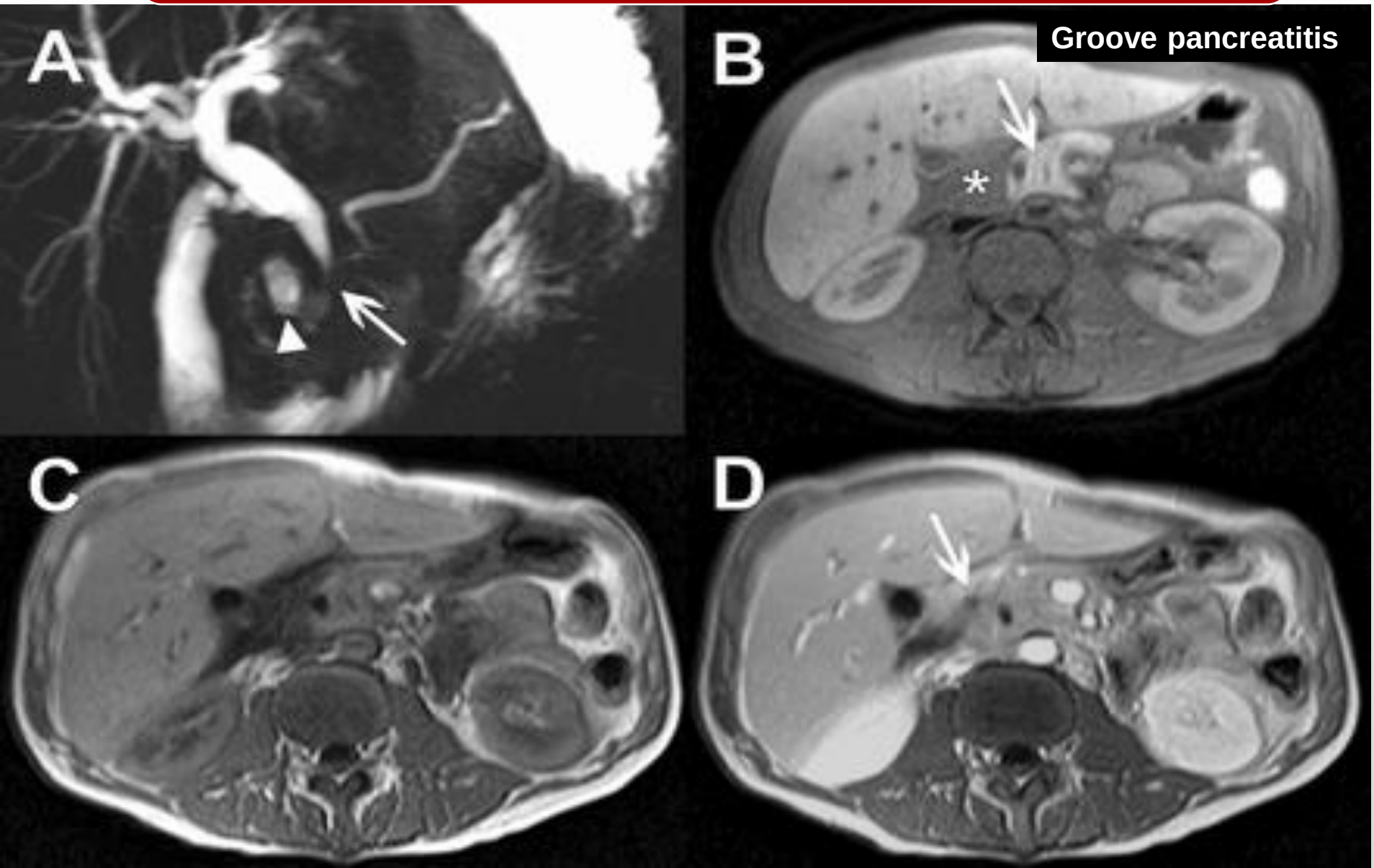
ERCP of normal pancreatic and biliary ducts.

LO4: ERCP

Endoscopic retrograde cholangiopancreatography (ERCP)



LO4: MRI



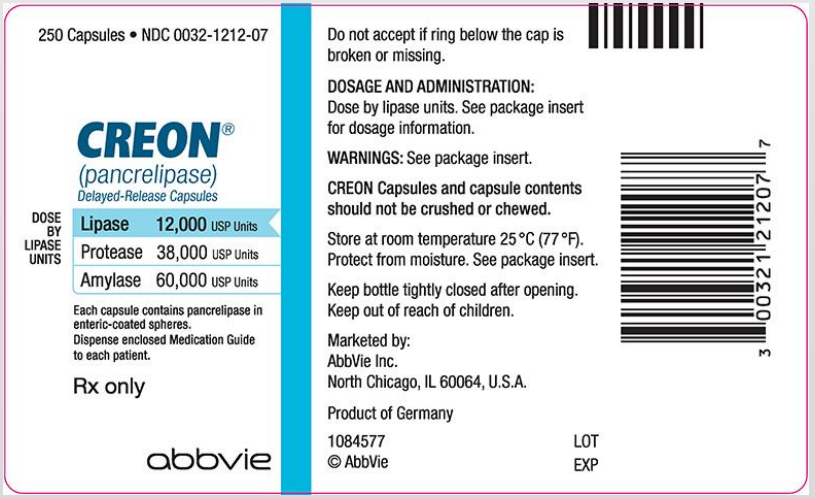
LO5: Treatment

- Aim - Pain control and management of maldigestion
- Pain
  - Avoid alcohol
  - Low fat meals
  - Antipain – narcotics tramadol, codeine(addiction)
  - Surgical pain control
    - Resection (local - - - - 95%) –causes pancreatic insufficiency
    - **Splanchinectomy, celiac ganglionectomy, nerve block**
  - Endoscopic treatment
    - Sphinctorotomy, dilatation of strictures, caliculi removal, duct stenting
      - Complications– acute pancreatitis, abscess, ductal damage, death
  - Pancreatic enzymes- Non enteric coated

Pancrelipase → CREON,Ultresa.

LO5: Treatment of maldigestion

- Pancreatic enzyme replacement
  - 2-3 enteric coated with meals
  - adjuvants with conventional tablets – H2 blockers, PPI, Na bicarbonate,
- Steatorrhea can be abolished if 10 % of normal lipase amount can be delivered to the duodenum at the right time.



LO6: Complications

Complications of chronic pancreatitis include:

- ***Pseudocyst formation***
- Fistula formation
- Pseudoaneurysms of large arteries close to the pancreas
- Stenosis of the common bile duct
- Splenic and/or portal venous obstruction

- Diabetes can develop in 70-90% of patients with chronic calcific pancreatitis