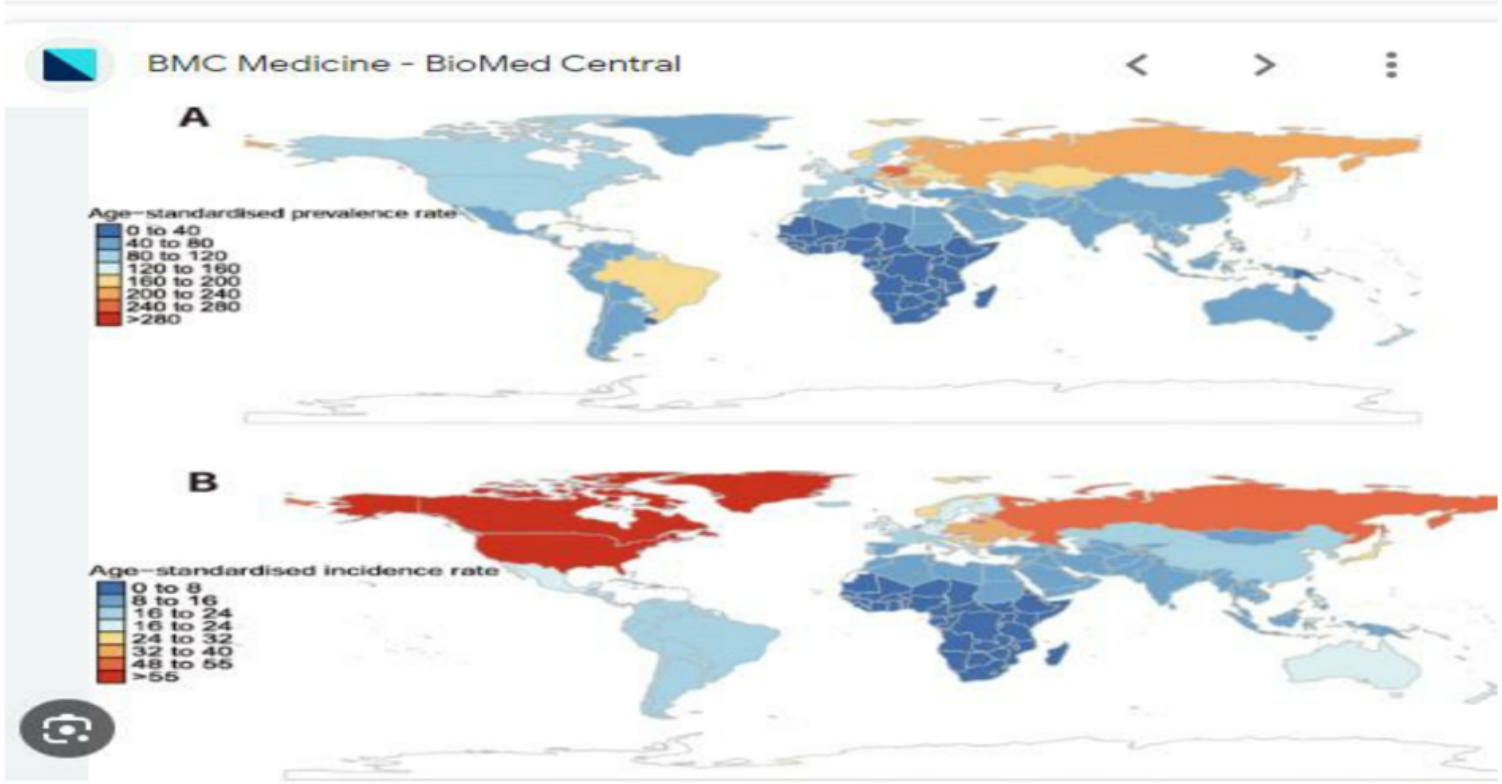

بسم الله الرحمن الرحیم

- Acute pancreatitis is an inflammatory condition of the pancreas characterized by abdominal pain and elevated levels of pancreatic enzymes in the blood

- The reported annual incidence of acute pancreatitis in the United States ranges from 4.9 to 35 per 100,000 population

- .The incidence of acute pancreatitis is increasing worldwid.e due to increased rates of obesity and gallstones



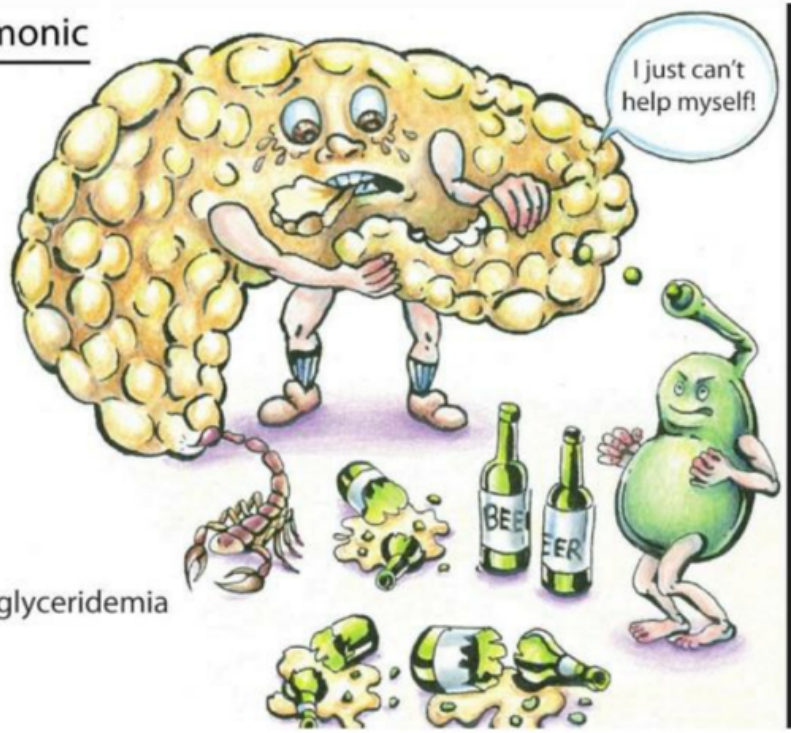
The global, regional, and national burden of pancreatitis in 195 countries and territories, 1990–2017: a systematic...

Visit

I GET SMASHED Mnemonic

Pancreatitis

Idiopathic
Gall Stones
Ethanol (Alcohol)
Trauma
Steroids
Mumps / **M**alignancy
Autoimmune
Scorpion Stings
Hypercalcemia / **H**ypertriglyceridemia
ERCP
Drugs



ETIOLOGY

- **Gallstones:** 40 to 70 percent of cases

- 3 to 7 percent of patients with gallstones develop pancreatitis

- risk of developing acute pancreatitis in patients with gallstones is greater in men

- stones with a diameter of less than 5 mm

ETIOLOGY

- **Alcohol** — approximately 25 to 35 percent

- **Hypertriglyceridemia** — Serum triglyceride concentrations above 1000 mg/Dl

- **ERCP** —
- 3 percent of patients undergoing diagnostic ERCP,
- 5 percent undergoing therapeutic ERCP,

ETIOLOGY

- **Genetic risk** — Patients with genetic risk for pancreatitis may present as
- recurrent
- Childhood
- progress to chronic pancreatitis.
- mutations in the *PRSS1* gene, which encodes cationic trypsinogen,
- Mutations in the *CFTR* gene
- mutations in the *SPINK1*, which may act as a disease modifier and lower the threshold for developing pancreatitis

ETIOLOGY

- **Medications**

- rare (<5 percent)

- prognosis is generally excellent

- mortality is low .

Mechanisms:

1-immunologic reactions (6-MP, aminosalicylates, sulfonamides)

2-direct toxic effect (diuretics, sulfonamides)

3-accumulation of a toxic metabolite (valproicacid, pentamidine, tetracycline)

4- ischemia (diuretics, azathioprine)

5-intravascular thrombosis (estrogen)

6-increased viscosity of pancreatic juice (diuretics and steroids)

ETIOLOGY

- **Rare causes**

1-Blunt or penetrating trauma

2Hypercalcemia

3-Infections and toxins

- **Viruses** – Mumps, coxsackievirus, hepatitis B, cytomegalovirus, varicella-zoster, herpes simplex, human immunodeficiency virus (HIV)
- ●**Bacteria** – *Mycoplasma, Legionella, Leptospira, Salmonella*
- ●**Fungi** – *Aspergillus*
- ●**Parasites** – *Toxoplasma, Cryptosporidium, Ascaris*

4-Anatomic or physiologic pancreatic anomalies

CLINICAL FEATURES

Pancreatitis symptoms

There are some general symptoms that may indicate acute / chronic pancreatitis, include:-

- Severe abdominal pain after having food
- Pain in upper abdominal pain that goes back
- Rapid heart beat
- Upset stomach
- Fever
- Unwanted weight loss
- Nausea and Vomiting
- Stinking and oily stool
- Tender and swollen belly
- Diarrhea
- Bleeding
- Dehydration

CLINICAL FEATURES

- PAIN:acute onset of persistent, severe epigastric and left upper quadrant abdominal pain .

- In patients with gallstone pancreatitis, the pain is well localized and the onset of pain is rapid, reaching maximum intensity in 10 to 20 minutes.

- In contrast, in patients with pancreatitis due to hereditary or metabolic causes or alcohol, the onset of pain may be less abrupt and the pain may be poorly localized.

- nausea and vomiting,.
- dyspnea due to diaphragmatic inflammation secondary to pancreatitis, pleural effusions, or ARDS

- Approximately 5 to 10 percent of patients with acute severe pancreatitis may have painless disease :

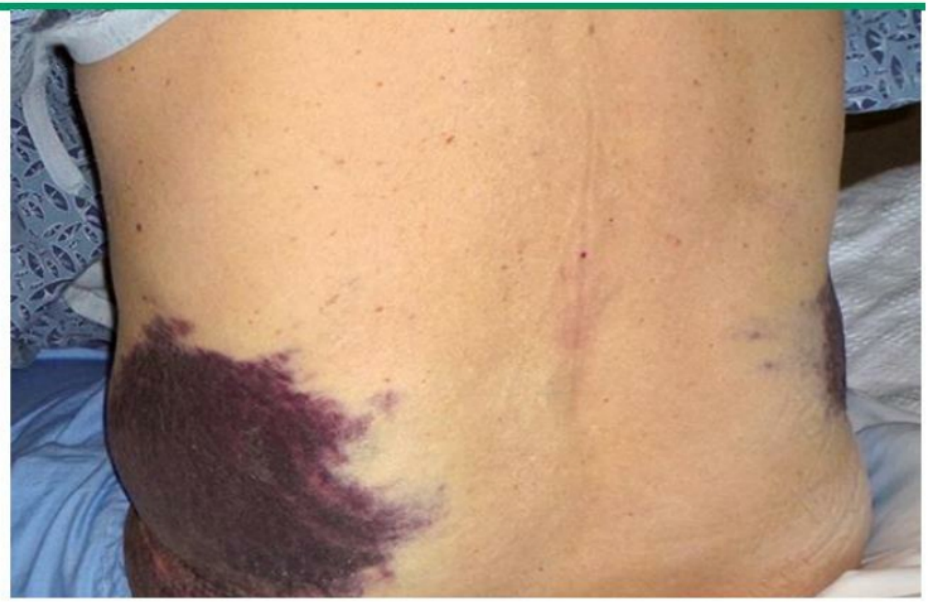
- postoperative and critically ill patients,
- patients on dialysis,
- organophosphate poisoning
- Legionnaire's disease

PHYSICAL EXAMINATION

- depending upon the severity of acute pancreatitis.
- significant tenderness to palpation
- hypoactive bowel sounds
- scleral icterus due to obstructive jaundice

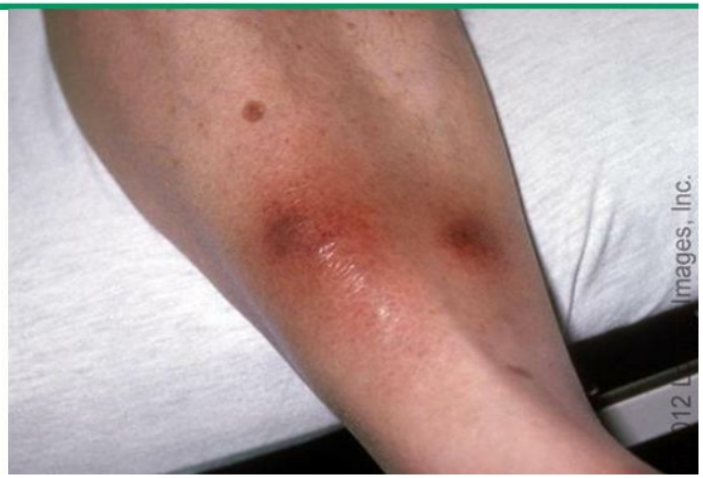
Grey Turner sign

Grey Turner sign



Pancreatic panniculitis

Pancreatic panniculitis



LABORATORY FINDINGS

- **Serum lipase**
- sensitivity 82 to 100 percent
- rises within 4-8hours of the onset of symptoms
- peaks at 24 hours

returns to normal within 8 to 14 days

elevations occur earlier and last longer as compared with elevations in amylase .

more sensitive as compared with amylase in patients with pancreatitis secondary to alcohol

LABORATORY FINDINGS

- **Serum amylase**

- Rises within 6 to 12 hours .
- Returns to normal within three to five days.
- Sensitivity 67 to 83 percent
- Specificity of 85 to 98 percent

- Acute pancreatitis is associated with elevations in

(CRP)

(IL)-6, IL-8, IL-10

(TNF)

PMN elastase

A CRP level above 150 mg/L at 48 hours is associated with severe pancreatitis.

IMAGING

- **Abdominal :**

unremarkable in mild disease

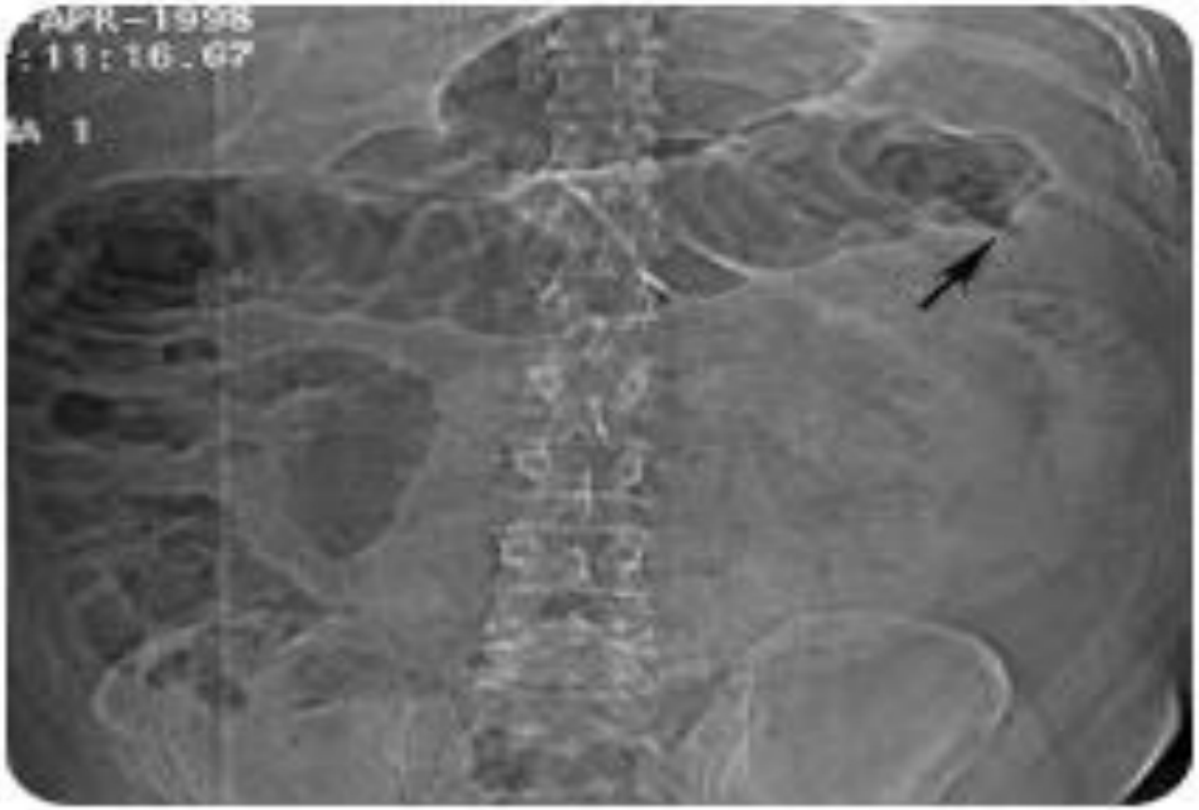
localized ileus of a segment of small intestine (sentinel loop)

colon cutoff sign in more severe disease.

A ground glass appearance may indicate the presence of an acute peripancreatic fluid collection.

chest radiographs :

- elevation of a hemidiaphragm, pleural effusions, basal atelectasis, pulmonary infiltrates, or ARDS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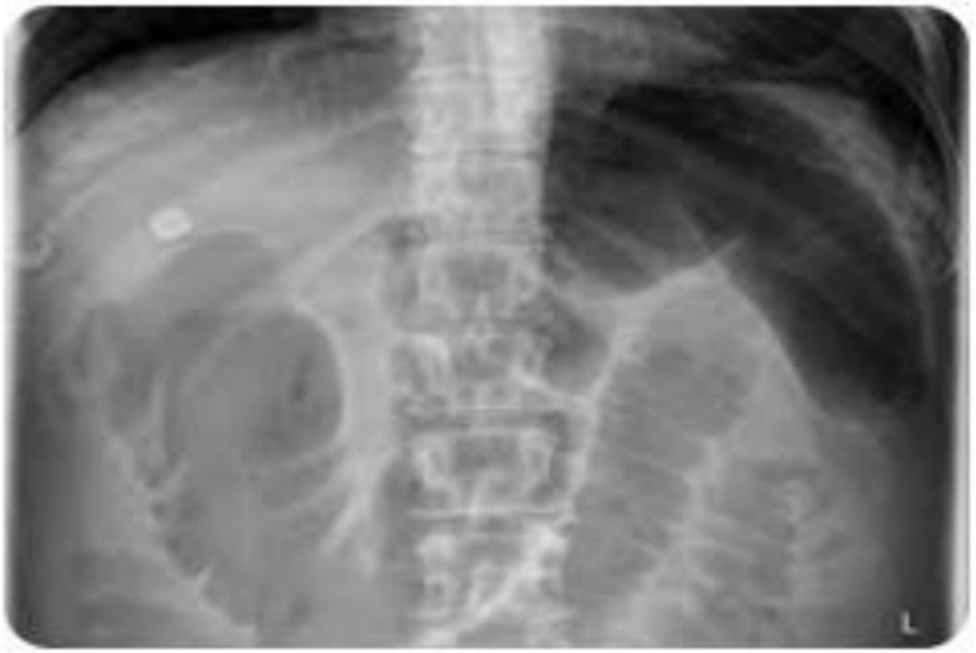
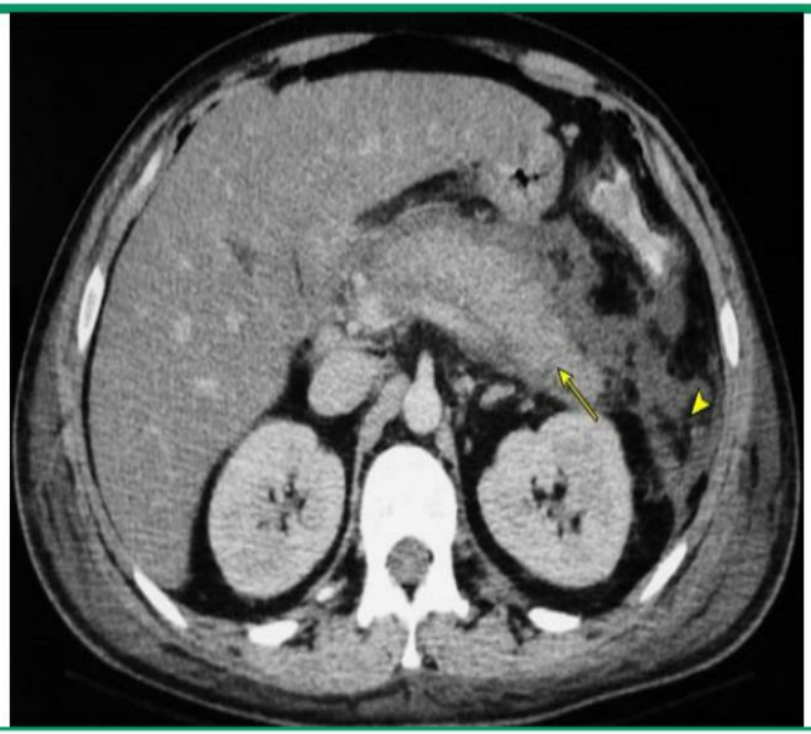


- **Abdominal ultrasound** —
- diffusely enlarged and hypoechoic
- Gallstones may be visualized in the gallbladder or the bile duct .

- **Abdominal computed tomography —**
- acute interstitial edematous pancreatitis
- include focal or diffuse enlargement of the pancreas
- Necrosis of pancreatic tissue

CT scan of necrotizing pancreatitis with peripancreatic necrosis

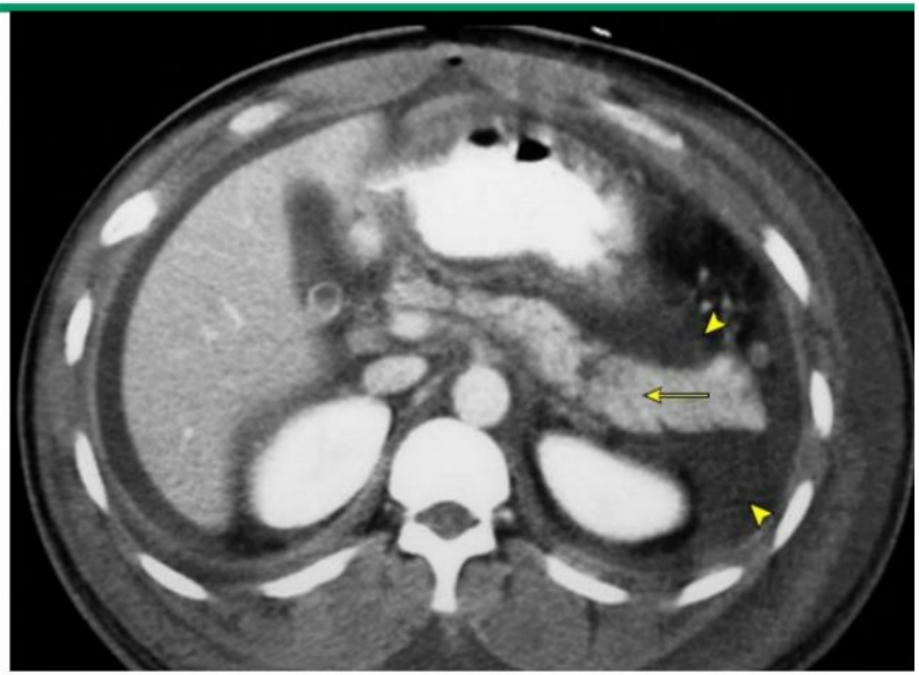


Computed tomography (CT) scan of a 34-year-old male with acute pancreatitis reveals the pancreas enhances homogeneously (arrow) but there is evidence of peripancreatic necrosis (arrowhead).

Graphic 88432 Version 1.0

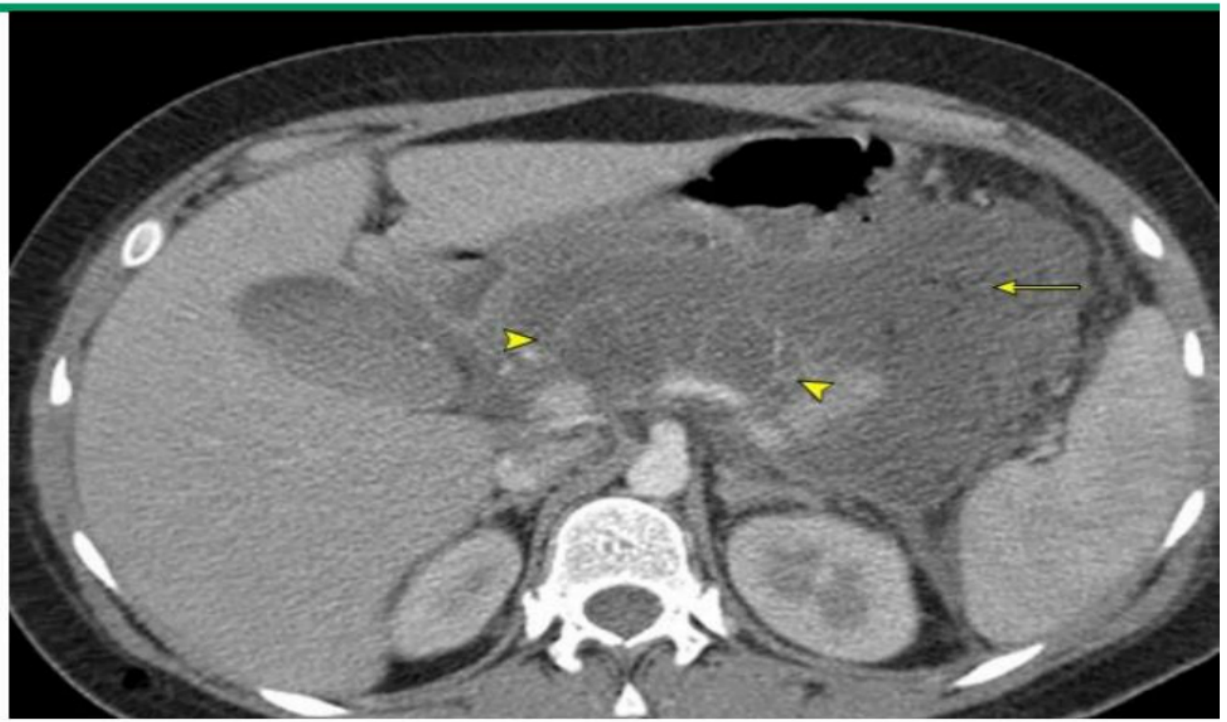
CT scan of acute interstitial pancreatitis with acute peripancreatic fluid collections

CT scan of acute interstitial pancreatitis with acute peripancreatic fluid collections



CT scan of acute necrotic collection

CT scan of acute necrotic collection



- **Magnetic resonance imaging —**
- diffuse or focal enlargement of the pancreatic gland
- margins of the pancreas may be blurred

- MRI has a higher sensitivity for the diagnosis of early acute pancreatitis as compared with CT scan and can better characterize the pancreatic and bile ducts and complications of acute pancreatitis .

DIAGNOSIS

- The diagnosis of acute pancreatitis requires the presence of two of the following three criteria:
- 1 acute onset of persistent, severe, epigastric pain often radiating to the back,
- 2elevation in serum lipase or amylase to three times or greater than the upper limit of normal,
- 3characteristic findings of acute pancreatitis on imaging CT or MRI or transabdominal ultrasonography.

- In patients with characteristic abdominal pain and elevation in serum lipase or amylase to three times or greater than the upper limit of normal, no imaging is required to establish the diagnosis of acute pancreatitis.

Diagnostic evaluation

- **Laboratory studies** — Elevation in serum lipase or amylase to three times or greater than the upper limit of normal is suggestive of acute pancreatitis.

- CBC, electrolytes, ALT, AST, bilirubin, calcium, triglyceride, albumin should be obtained to identify the cause and also rule out other causes of acute abdominal pain.
- A pregnancy test should be performed in all females of childbearing age.)

Diagnostic evaluation

- **Imaging**

- The presence of focal or diffuse enlargement of the pancreas on contrast-enhanced abdominal CT or MRI is suggestive of acute pancreatitis.

DIFFERENTIAL DIAGNOSIS

- Peptic ulcer disease
- Choledocholithiasis or cholangitis
- Cholecystitis
- Perforated viscus
- Intestinal obstruction
- Mesenteric ischemia

Disease course

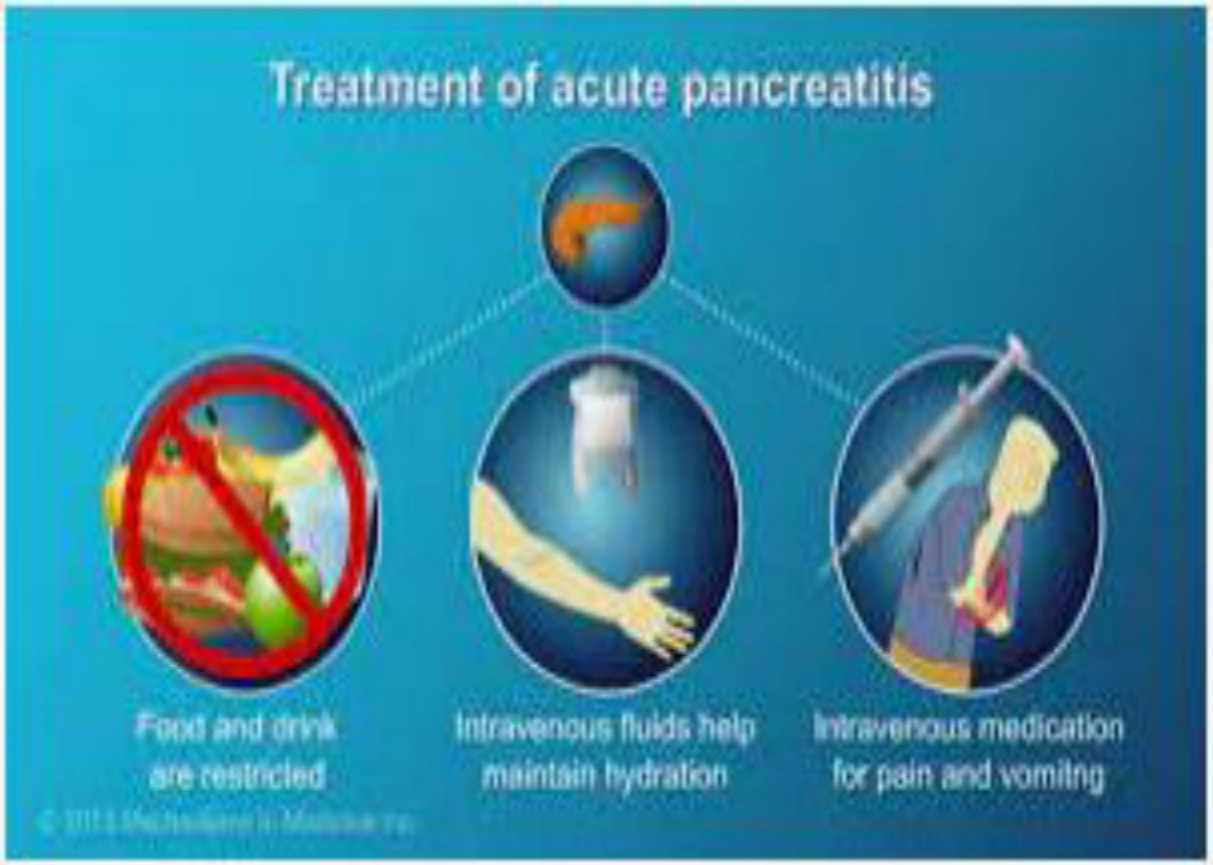
- Approximately 75 to 80 percent of patients with acute pancreatitis have acute interstitial edematous pancreatitis.

- Approximately 15 to 25 percent of patients have necrotizing pancreatitis with necrosis of the pancreatic parenchyma
- In most patients with acute pancreatitis, the disease is mild in severity and patients recover in three to five days without complications or organ failure.

Disease course

- approximately 20 percent of patients have moderately severe or severe acute pancreatitis with local or systemic complications or organ failure
- . The overall mortality in acute pancreatitis is under 5 percent,

- interstitial pancreatitis as compared with those with necrotizing pancreatitis (3 versus 17 percent.


Treatment of acute pancreatitis
Food and drink are restricted
Intravenous fluids help maintain hydration
Intravenous medication for pain and vomiting

MANAGEMENT

- **CLASSIFICATION —**

- Mild :absence of organ failure and local or systemic complications
- Moderately :which is characterized by no organ failure or transient organ failure (<48 hours) and/or local complications
- Severe : which is characterized by persistent organ failure (>48 hours) that may involve one or multiple organs

Modified Marshall scoring system for organ dysfunction

Modified Marshall scoring system for organ dysfunction

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO_2/FiO_2)	>400	301-400	201-300	101-200	≤101
Renal*					
(serum creatinine, micromol/L)	≤134	134-169	170-310	311-439	>439
(serum creatinine, mg/dL)	<1.4	1.4-1.8	1.9-3.6	3.6-4.9	>4.9
Cardiovascular (systolic blood pressure, mmHg)¶	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH <7.3	<90, pH <7.2
For nonventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen (L/min)	**FiO_2 (percent)**				
Room air	21				
2	25				
4	30				
6-8	40				
9-10	50				

Indications for monitored or intensive care

- 1-Patients with severe acute pancreatitis
- 2-Patients with acute pancreatitis and one or more of the following parameters:
- •Pulse <40 or >150 beats/minute
- •Systolic arterial pressure <80 mmHg or mean arterial pressure <60 mmHg or diastolic arterial pressure >120 mmHg
- •Respiratory rate >35 breaths/minute
- •Serum sodium <110 mmol/L or >170 mmol/L
- •Serum potassium <2.0 mmol/L or >7.0 mmol/L
- •PaO_2 <50 mmHg
- •pH <7.1 or >7.7
- •Serum glucose >800 mg/dL
- •Serum calcium >15 mg/dL
- •Anuria
- •Coma

Fluid replacement

- Timing?
- Rate?
- Type of fluid?
- Duration of treatment?
- Appropriate goal?

- 1.5 mL/kg/hour with a 10 mL/kg bolus

- reassessed at frequent intervals in the first six hours of admission and for the next 24 to 48 hours

- The rate of fluid resuscitation is adjusted based on clinical assessment, hematocrit, (BUN), creatinine values .
-

fluid replacement can be assessed

- improvement in vital signs (goal heart rate <120 beats/minute, mean arterial pressure between 65 to 85 mmHg),
- urine output (>0.5 to 1 cc/kg/hour)

- reduction in hematocrit (goal 35 to 44 percent)

- BUN over 24 hours

- There is conflicting evidence that fluid resuscitation with lactated Ringer's solution can reduce the incidence of (SIRS) as compared with normal saline .

- In rare patients with acute pancreatitis due to hypercalcemia, lactated Ringer's is contraindicated because it contains 3 mEq/L calcium.

Pain control

- ●Attention to adequate fluid resuscitation should be the first priority in addressing abdominal pain, as hypovolemia from vascular leak and hemoconcentration can cause ischemic pain and resultant lactic acidosis
- ●Opioids are safe and effective at providing pain control in patients with acute pancreatitis

Monitoring

Vital signs

Urine output

Electrolytes

Serum glucose levels

abdominal compartment syndrome

Nutrition

- **Oral**
- In the absence of ileus, nausea or vomiting, oral feeding can be initiated early (within 24 hours) as tolerated, if the pain is decreasing and inflammatory markers are improving .
- low residue, low fat, soft diet,
- patients require enteral if they cannot tolerate oral diet by day five
- In a systematic review of 11 randomized trials that included 948 patients with acute pancreatitis, early refeeding (≤48 after hospitalization), as compared with delayed refeeding, did not increase adverse effects or exacerbate symptoms

- **Enteral**
- Radiologic or endoscopic placement of a jejunal feeding tube beyond the ligament of Treitz. If placement of a nasojejunal feeding tube is not possible, nasogastric feeding should be initiated

- high protein, low fat, semi-elemental feeding formulas

Advantage of enteral nutrition

- maintain the intestinal barrier
- prevents bacterial translocation from the gut
- avoidance of the complications associated with parenteral nutrition including those secondary to venous access and blood stream infections.

- Signs that the formula is not tolerated include:
- increased abdominal pain, vomiting (with nasogastric feeding), bloating, diarrhea (>5 watery stools or >500 mL per 24 hours

Antibiotics

Up to 20 percent of patients with acute pancreatitis develop an extrapancreatic infection (eg, bloodstream infections, pneumonia, and urinary tract infections.

Extrapancreatic infections are associated with an increase in mortality .

When an infection is suspected, antibiotics should be started while the source of the infection is being determined.

if cultures are negative and no source of infection is identified, antibiotics should be discontinued.

MANAGEMENT OF UNDERLYING PREDISPOSING CONDITIONS

- **Gallstone pancreatitis:**
- ERCP**?**
- urgent (<24 hours) ERCP?
- EUS or MRCP**?**
- Cholecystectomy **?**
- mild pancreatitis, cholecystectomy can usually be performed safely within seven days after recovery

Chronic Pancreatitis: Diagnosis and Management

- **Recentdefinition**

  •• pathologic fibro-inflammatory syndrome of the pancreas in individuals with genetic, environmental, and/or other risk factors

- **Definition**
  - • Chronic pancreatitis can result from episodes of acute pancreatitis of any cause, most commonly in those with multiple relapsing episodes of acute pancreatitis
  - • Acute and chronic pancreatitis can best be thought of as a continuum and two parts of the same spectrum of disease rather than as two completely separate conditions

- **Abdominalpain**
  - •• Abdominal pain is the most common clinical symptom in c hronic pancreatitis.
  - •• Epigastric region and often radiates to the back. It may be worse when recumbent and patients may experience postprandial exacerbation.
- • Pain is most common in those with chronic pancreatitis due to alcohol or tobacco use and in those with idiopathic pancreatitis occurring at a young age.
  - •• Nausea, vomiting, and anorexia are

- **Steatorrhea**

  •• Oily or floating stool due to fat maldigestion but may not have

  frequent or watery diarrhea.

  •• Approximately 90 percent of pancreatic exocrine secretory capacity

  must be lost

  •• Long-standing chronic pancreatitis (usually >5 to 10 years)

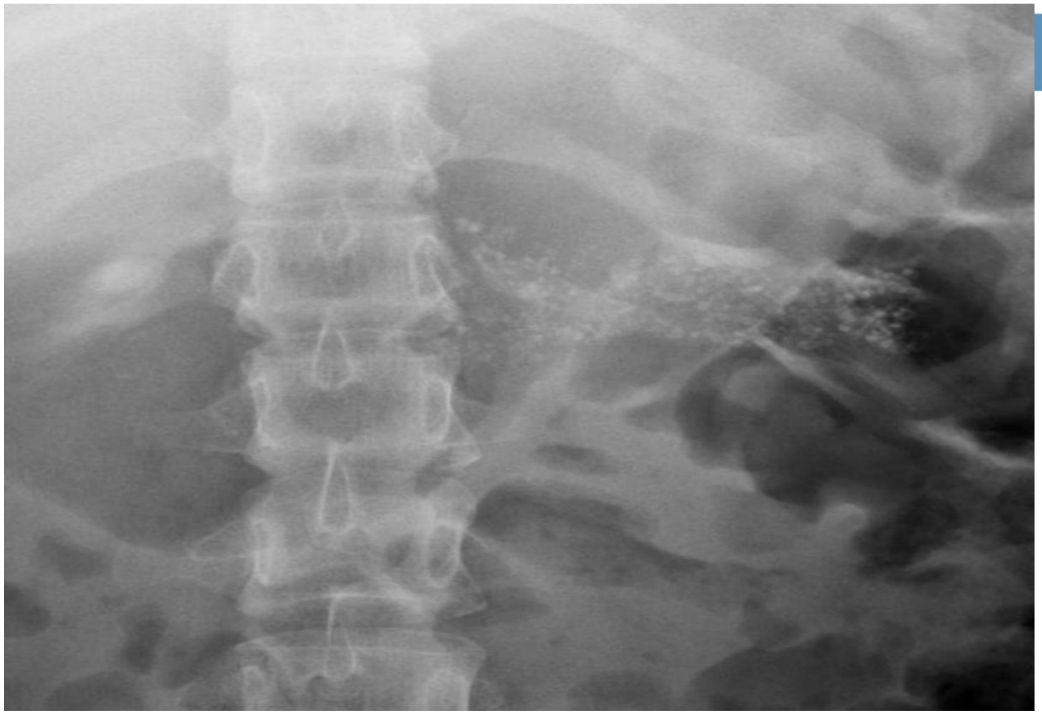
- **Labs**
  - • Peak levels of elevation of amylase and lipase tend to decrease with each additional pain flare and who are evolving towards chronic pancreatitis
  - •• Deficiencies of fat-soluble vitamins frequently develop, with vitamin D
  - •in particular, which increases the risk of metabolic bone disease
  - • Although triglyceride levels above 1000 mcg/mL are typically needed to produce the first episode of acute pancreatitis in these

Approach

- Suspected in patients with chronic abdominal pain and/or a history of relapsing acute pancreatitis, symptoms of pancreatic exocrine (diarrhea, steatorrhea, or weight loss) insufficiency, or pancreatogenic diabetes

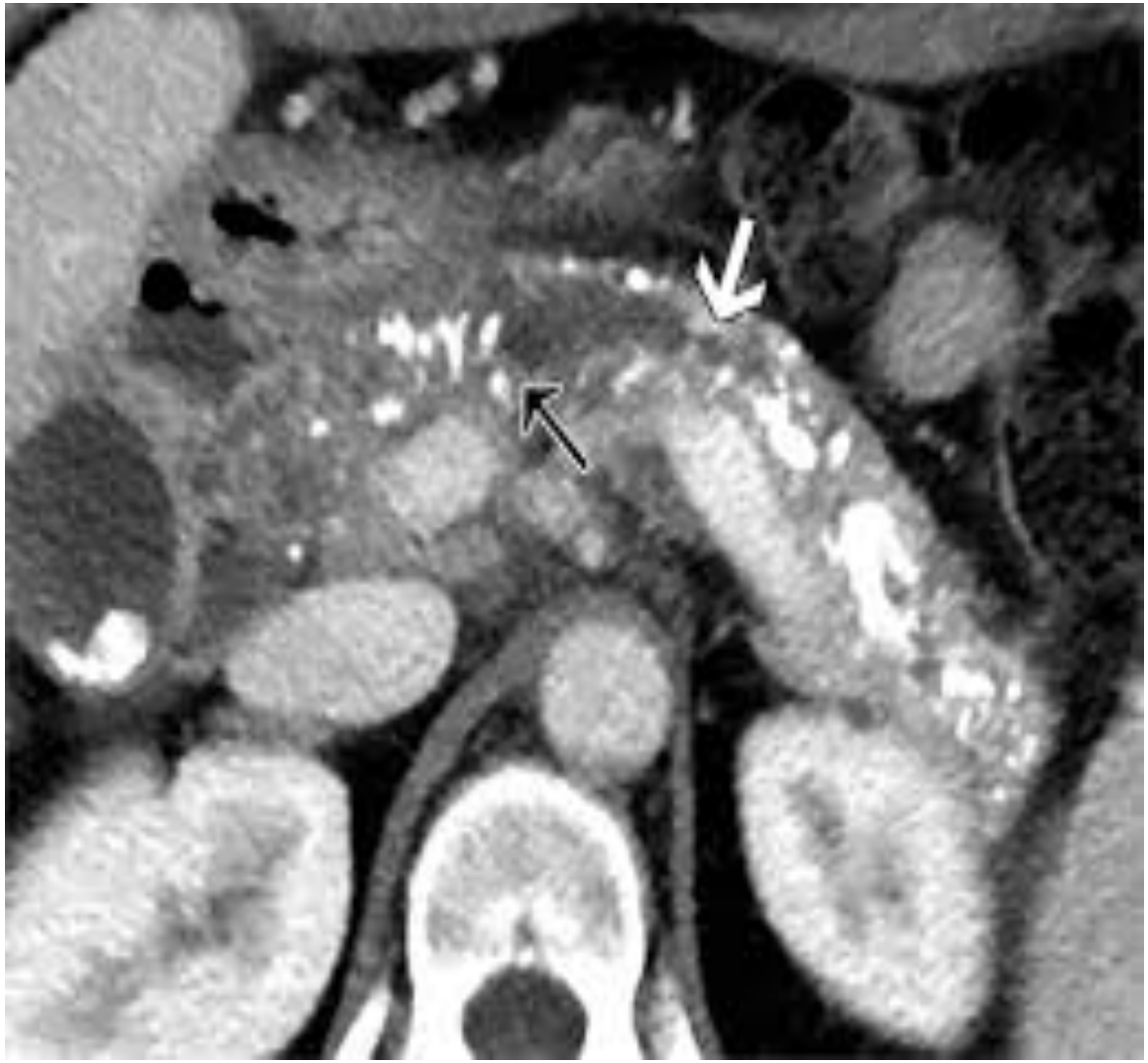
•• CT,MRI,MRCP

- • In patients with suspected chronic pancreatitis but equivocal cross- sectional imaging, we perform either a direct pancreatic function test with secretin, if available, or endoscopic ultrasound (EUS)



- **CTscan**
  - Atrophy of the pancreas, ductal dilatation, and multiple parenchymal

    and intraductal calcifications
  - Sensitivity ranges from 80 to 90 percent, with a specificity of approximately 85 percent



Diagnosis By EUS Criteria

Parenchymal

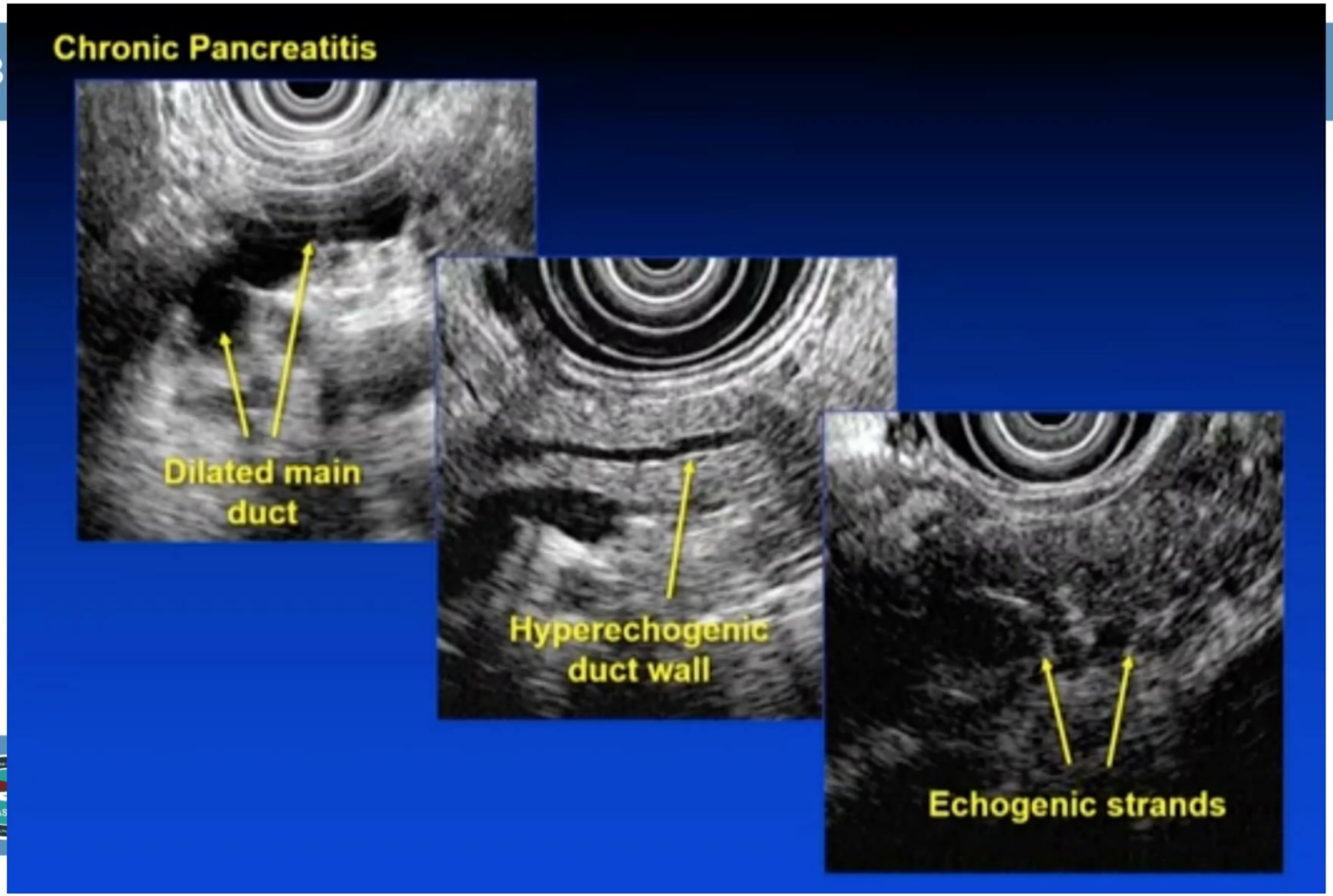
- Hyperechoic foci
- Hyperechoic strands
- Lobularity of contour
- Cysts
- Calcification

Ductal (more predictive)

- Main duct dilation
- Main duct irregularity
- Hyperechoic duct walls
- Visible side branches
- Stones

3 criteria: mild chronic pancreatitis
4-5 criteria: moderate chronic pancreatitis
>5 criteria: severe pancreatitis

Sensitivity 93%, specificity 94%


Chronic Pancreatitis
Dilated main duct
Hyperechogenic duct wall
Echogenic strands

- **ACG2020**

  •• We recommend computed tomography (CT) or MRI for the first-line

- • EUS, because of its invasiveness and lack of specificity, should be used only if the diagnosis is in question after cross-sectional imaging is performed

- • We suggest performing s-MRCP when the diagnosis of CP following cross-sectional imaging or EUS is not confirmed and the clinical suspicion remains high

- **Indirectpancreaticfunctiontesting**

  •• Fecal elastase measurement is performed on a random stool sample,

    •which should be solid or semisolid.

  •• The assay does not actually measure elastase-1 but instead measures

    •chymotrypsin-like elastases.

- • Levels <100 mcg/g stool are reasonably

- **ACG2020**
  - • Pancreatic function testing is an important means of diagnosing exocrine pancreatic insufficiency; however, its role in establishing the diagnosis of CP is complementary.

- **ETIOLOGY AND RISK FACTORS**
  - "TIGAR-O"
  - Toxic-metabolic: alcohol, smoking, TG, DM
  - Idiopathic
  - Genetic: PRSS1
  - Autoimmune: types 1 and 2
  - Recurrent and severe acute pancreatitis
  - Obstructive

- **ACG2020**

  •• Identification of the disorders(s) underlying pancreatic inflammation

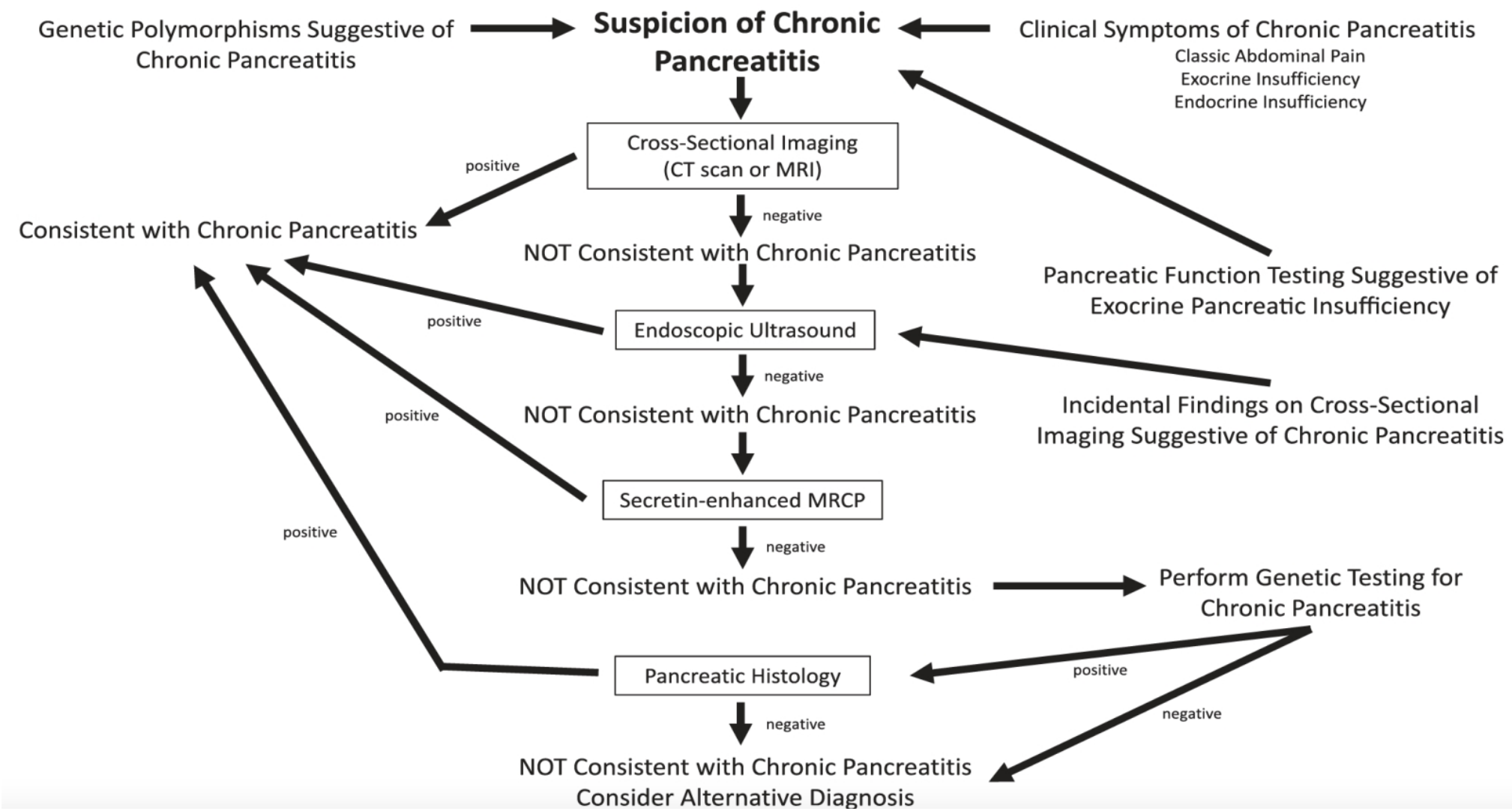
  •is important in predicting progression to CP.

- • The development of DM in CP is most likely related to duration of disease, although other etiologic factors such as BMI and smoking status may incur an increased risk.

- • There is a lack of evidence to suggest that performing screening examinations on patients with CP to detect pancreatic malignancy is beneficial.

- Genetictesting
  - •• **One** or more of the following criteria:
  - •• •An unexplained documented episode of acute pancreatitis as a child
  - •• •Recurrent acute attacks of pancreatitis for which there is no identifiable cause
  - • •Idiopathic chronic pancreatitis, particularly when the onset of acute pancreatitis occurs before age 25 or in patients with chronic pancreatitis at a young age (<40 years)
  - •• •A family history of recurrent acute pancreatitis, idiopathic chronic

- **ACG2020**
  - • We recommend genetic testing in patients with clinical evidence of a pancreatitis-associated disorder or possible CP in which the etiology is unclear, especially in younger patients


Genetic Polymorphisms Suggestive of Chronic Pancreatitis
Suspicion of Chronic Pancreatitis
Clinical Symptoms of Chronic Pancreatitis
Classic Abdominal Pain
Exocrine Insufficiency
Endocrine Insufficiency
Cross-Sectional Imaging (CT scan or MRI)
positive
Consistent with Chronic Pancreatitis
negative
NOT Consistent with Chronic Pancreatitis
Pancreatic Function Testing Suggestive of Exocrine Pancreatic Insufficiency
positive
Endoscopic Ultrasound
negative
NOT Consistent with Chronic Pancreatitis
Incidental Findings on Cross-Sectional Imaging Suggestive of Chronic Pancreatitis
positive
Secretin-enhanced MRCP
negative
positive
NOT Consistent with Chronic Pancreatitis
Perform Genetic Testing for Chronic Pancreatitis
Pancreatic Histology
positive
negative
negative
NOT Consistent with Chronic Pancreatitis
Consider Alternative Diagnosis

- **Management**

  •• **Cessation of alcohol and tobacco**

  •• **Diet and supplements:** low- to moderate-fat meals, high protein

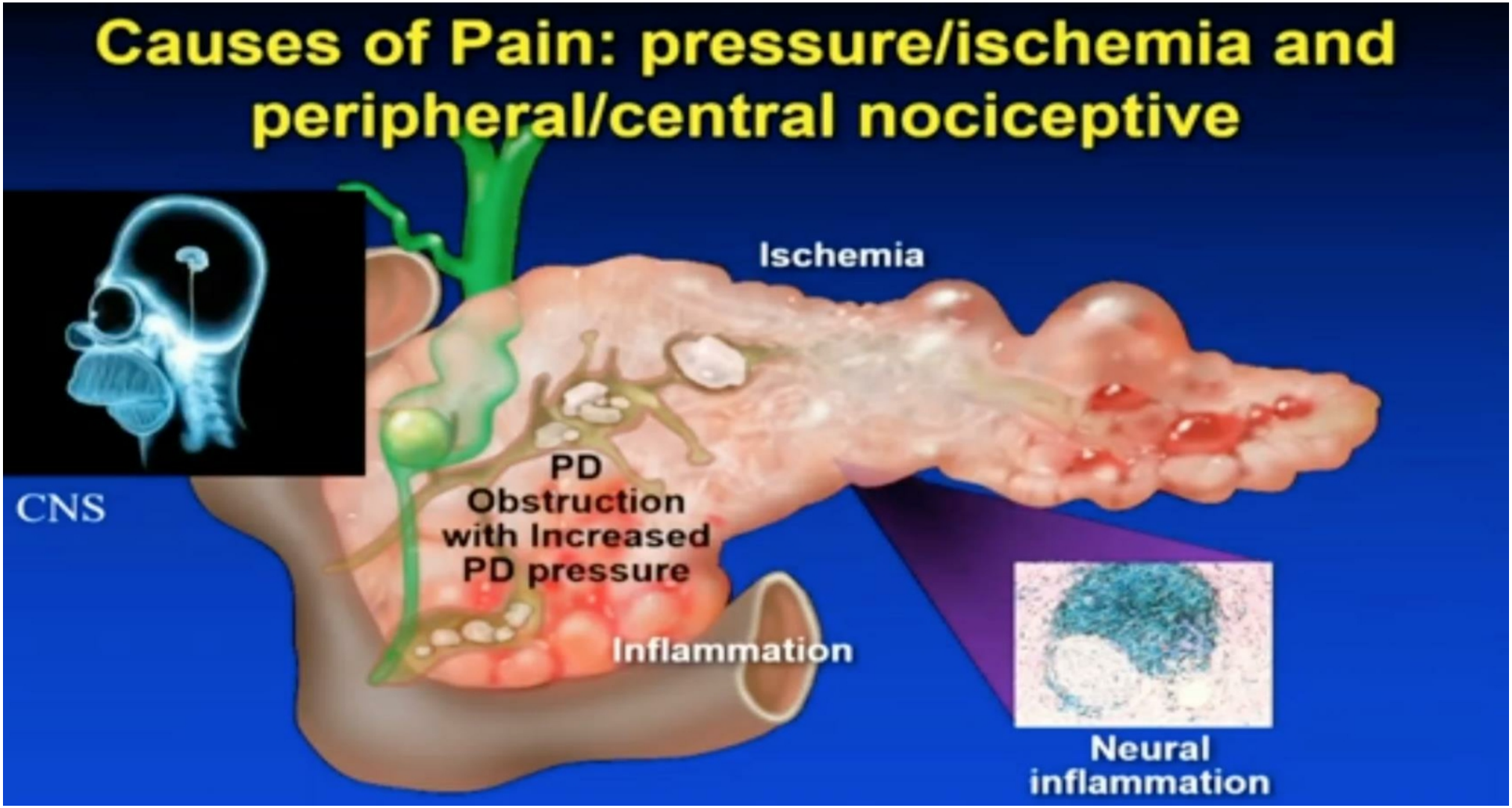
  •foods, small meals, and avoid dehydration.

  •• Vitamin D and calcium supplementation should be nearly universal

- **MANAGEMENTOFPAIN**

- • R/O other reasons (peptic ulcer disease, superimposed pancreatic carcinoma, narcotic bowel, gastroparesis, pancreatic pseudocyst, duodenal or biliary obstruction)

•• Pain is worse within 5 to 10 minutes of eating as a result of stimulating the inflamed gland


Causes of Pain: pressure/ischemia and peripheral/central nociceptive
CNS
Ischemia
PD
Obstruction
with Increased
PD pressure
Inflammation
Neural
inflammation

- **Analgesics**

  •• Initial: (NSAIDs) and acetaminophen

  •• Minimize opioids, especially chronic narcotic use: pain control, rather than total absence of pain

  •• Lower-potency opioid agents like tramadol

- • Add adjunctive agents including TCAs, SSRIs, and SNRIs (eg, duloxetine) or gabapentoids (pregabalin or gabapentin

- **Antioxidantsandothertherapies**
  - Combination of vitamin E (200 international units [IU]),
  - Vitamin C (500 mg),
  - Beta-carotene (5000 IU),
  - Selenium (500 mcg), and
  - Methionine (1000 mg

• Pancreaticenzymes

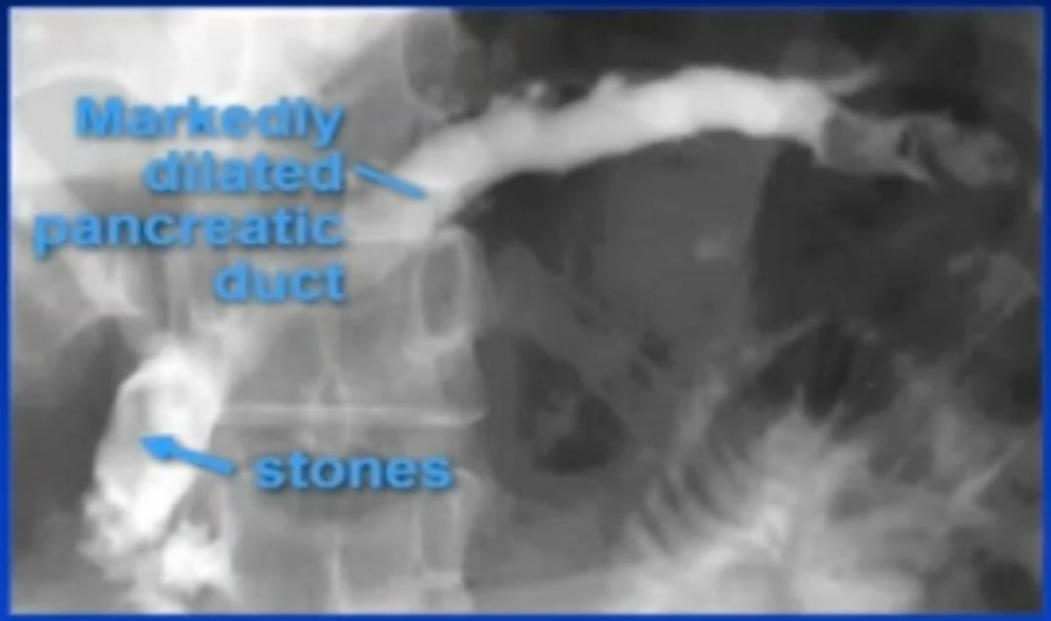
- • Pancreatic enzyme supplementation may reduce pain due to **abdominal cramping and diarrhea** due to exocrine pancreatic insufficiency in selected patients

•• Unclear whether enzyme supplementation treats pain secondary to

•pancreatic inflammation or ductal stones

•• Only the non-enteric preparations release the majority of their proteases in the duodenum, and these agents are not easily available

Endoscopic Approaches for Chronic Pancreatitis



- Papillotomy
- Stenting
- Endoscopic stone removal
- Lithotripsy
- Combined therapies

PD Strictures: Approach

- Predilation
- Stent placement (?multiple)
- **Planned vs on demand stent exchange/addition (4-5 procedures over a year)**
- Stent-free trial
- Complete stricture resolution may not be necessary for relief
- Re-intervention or referral to surgery as needed

PD Stones: Approach

- Amenable:
  - MPD stone in the head
  - Upstream dilation
- Not amenable
  - Extensive stone burden
    - Adherent
    - impacted
  - Stones behind strictures
  - Complex or long strictures
  - Stones in the tail

- **Celiacplexusblock**

  •• Unclear which patients will derive the most benefit and the pain relief is transient, lasting for three to six months

- • "as-needed" basis, generally separating procedures by three months or more if the patient has had clinical benefit from the initial celiac intervention

  •• By (EUS) guidance, which is safer and more effective than CT.

  •• Anesthetic (typically bupivacaine)

- **Surgicalresection**
  - Small-diameter main pancreatic duct (<6 to 7 mm) with refractory pain
  - Directed partial resection
  - **Pancreatoduodenectomy (Whipple operation)**
  - **Duodenum-preserving pancreatic head resection (DPPHR)**
  - **Total pancreatectomy with islet cell autotransplantation (TPIAT)**

- **ACG2020**

  •• We recommend alcohol cessation in patients with CP

  •• We recommend smoking cessation in patients with CP

- • We recommend surgical intervention over endoscopic therapy in patients with obstructive CP for the long-term relief of pain if first-line endoscopic approaches to pancreatic drainage have been exhausted or unsuccessful

  •• We suggest considering the use of antioxidant

- **ACG2020**
  - We do **not** suggest the use of pancreatic enzyme supplements to improve pain in CP (conditional recommendation, low quality of evidence).
  - We suggest considering celiac plexus block for treatment of pain in CP

- **MANAGEMENTOFPANCREATIC**
- **INSUFFICIENCY**
  - Late manifestation of chronic pancreatitis, usually occurring after
    - more than five years of disease
  - **Nutritional assessment**
  - anthropometric (body mass index, muscle mass or strength) and
    - nutritional assessment
  - Laboratory testing for vitamin deficiency should be performed at
    - baseline and annually thereafter

- Serum retinol and retinol-binding protein
- • Serum 25-hydroxyvitamin D (25[OH]D)
- • Serum alpha-tocopherol levels
- • International normalized ratio (INR)
- • Prealbumin
- • B12
- • HgBA1c to assess for concomitant pancreatogenic diabetes
- • Baseline (DEXA) scan every one to two years

- **Pancreaticenzymereplacement therapy**

•• Mild exocrine pancreatic insufficiency is 25,000 to 50,000 USP units

- (500 units of lipase per kg body weight per meal) taken with the first bite of each main meal

•• One-half that amount with snacks

•• Moderate insufficiency (most patients), we typically use 75,000 to

•100,000 lipase units with each meal.

- **Pancreatogenicdiabetes**
  - • Metformin is the preferred oral hypoglycemic agent as there is circumstantial evidence that it may lower the risk of secondary pancreatic carcinoma
  - •• Insulin is often needed
  - • (GLP)-1 analogues and (DPP4) inhibitors have not been well studied in patients with chronic pancreatitis and are generally avoided due to their risk of acute pancreatitis