

Pancreatitis

Dr. Amitesh Aggarwal

Pancreatic inflammatory disease - **classified** as:

Acute pancreatitis

Chronic pancreatitis

The pathologic spectrum

- Interstitial pancreatitis - mild and self-limited



- Necrotizing pancreatitis - extent of pancreatic necrosis correlate with severity of attack and systemic manifestations

Causes of Acute Pancreatitis

Common Causes

Gallstones (mirizzi's chiasis)

2

(15-30%)

Alcohol

ERCP

Trauma (blunt abdominal)

Postoperative

Drugs

Sphincter of Oddi dysfunction

Hypertriglyceridemia

1

(30-60%)

Uncommon Causes

vascular causes & vasculitis

CTD and TTP

Cancer of the pancreas

Hypercalcemia

Periampullary diverticulum

Pancreas divisum

pancreatitis

levels are usually >1000 mg/dL

systemic sclerosis, renal failure

Rare Causes

Infections (mumps, coxsackievirus, CMV, echovirus, parasites)

Autoimmune (e.g., Sjögren's syndrome)

Drugs

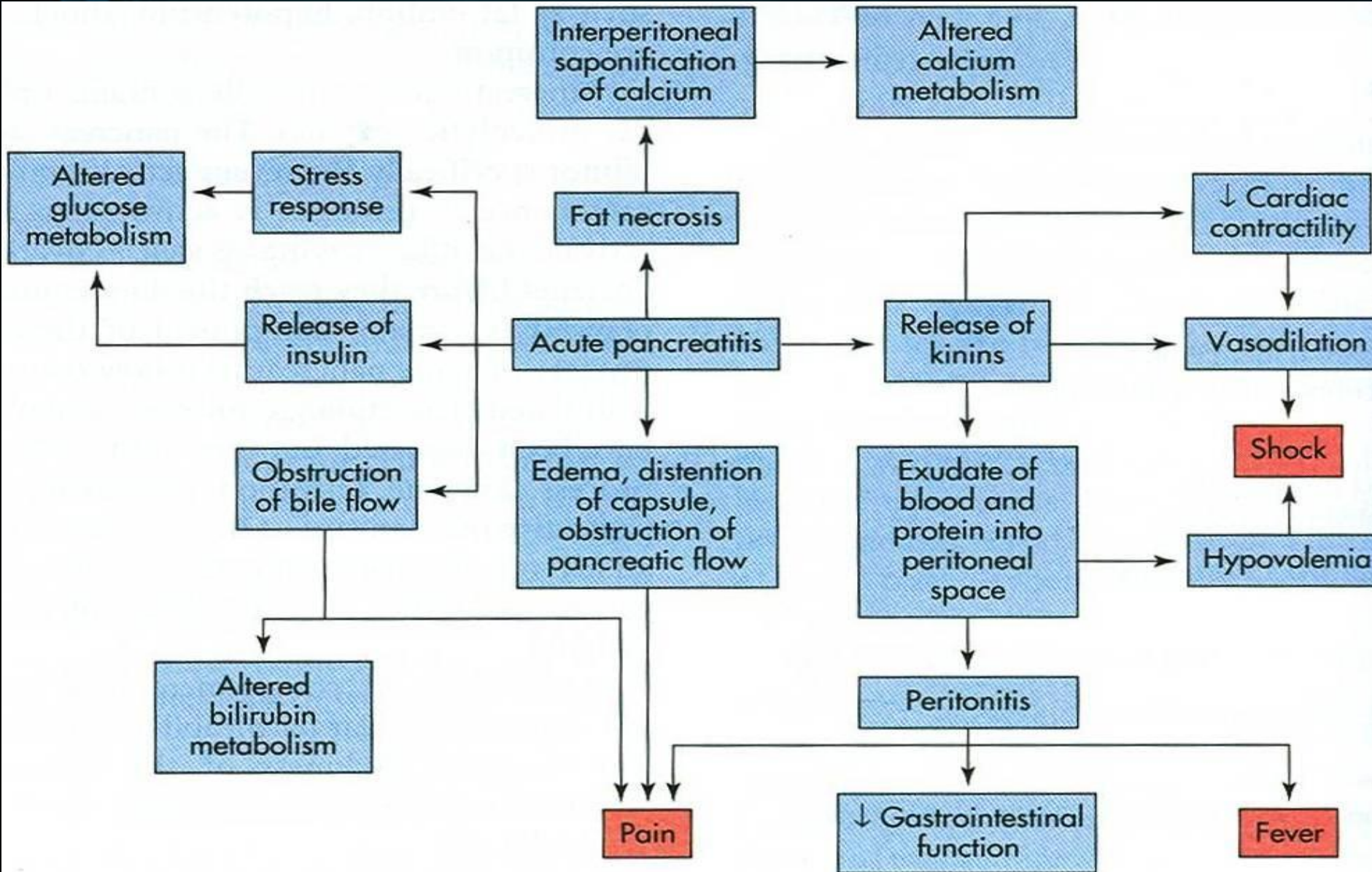
(Definitely associated with acute pancreatitis)

- Azathioprine
- Sulfonamides
- Tetracycline
- Valproic acid
- Didanosine
- Methyldopa
- Estrogens
- Furosemide
- 6-Mercaptopurine
- Pentamidine
- 5-aminosalicylic acid compounds
- Corticosteroids
- Octreotide

Pathophysiology

- Autodigestion is a currently accepted pathogenic theory
- Proteolytic enzymes and lipolytic enzymes are activated in the pancreas rather than in the intestinal lumen

Pathophysiology



Clinical Features



- **Abdominal pain** - major symptom
- May vary from mild to severe, constant, incapacitating distress
- Pain, which is steady and boring in character, is located in epigastrium and periumbilical region and often radiates to back, chest, flanks, and lower abdomen
- Frequently more intense when patient is supine
- Nausea, vomiting, abdominal distention also frequent

Clinical Features

- Distressed and anxious patient
- Low-grade fever, tachycardia, hypotension
- Shock not unusual and may result from (1) hypovolemia (2) increased release of kinin peptides (3) systemic effects of proteolytic and lipolytic enzymes
- Jaundice occurs infrequently
- Erythematous skin nodules due to subcutaneous fat necrosis
- Pulmonary findings, including basilar rales, atelectasis, pleural effusion (L>R)
- Abdominal tenderness and muscle rigidity
- Bowel sounds are usually diminished or absent

Clinical Features

- An enlarged pancreas with walled off necrosis or a pseudocyst may be palpable in the upper abdomen later (i.e., four to six weeks)
- Cullen's sign - faint blue discoloration around umbilicus , result of hemoperitoneum
- Turner's sign - blue-red or green-brown discoloration of the flanks, reflects tissue catabolism of hemoglobin



Traditional severity indices

- Atlanta criteria
- Ranson criteria
- APACHE II Score

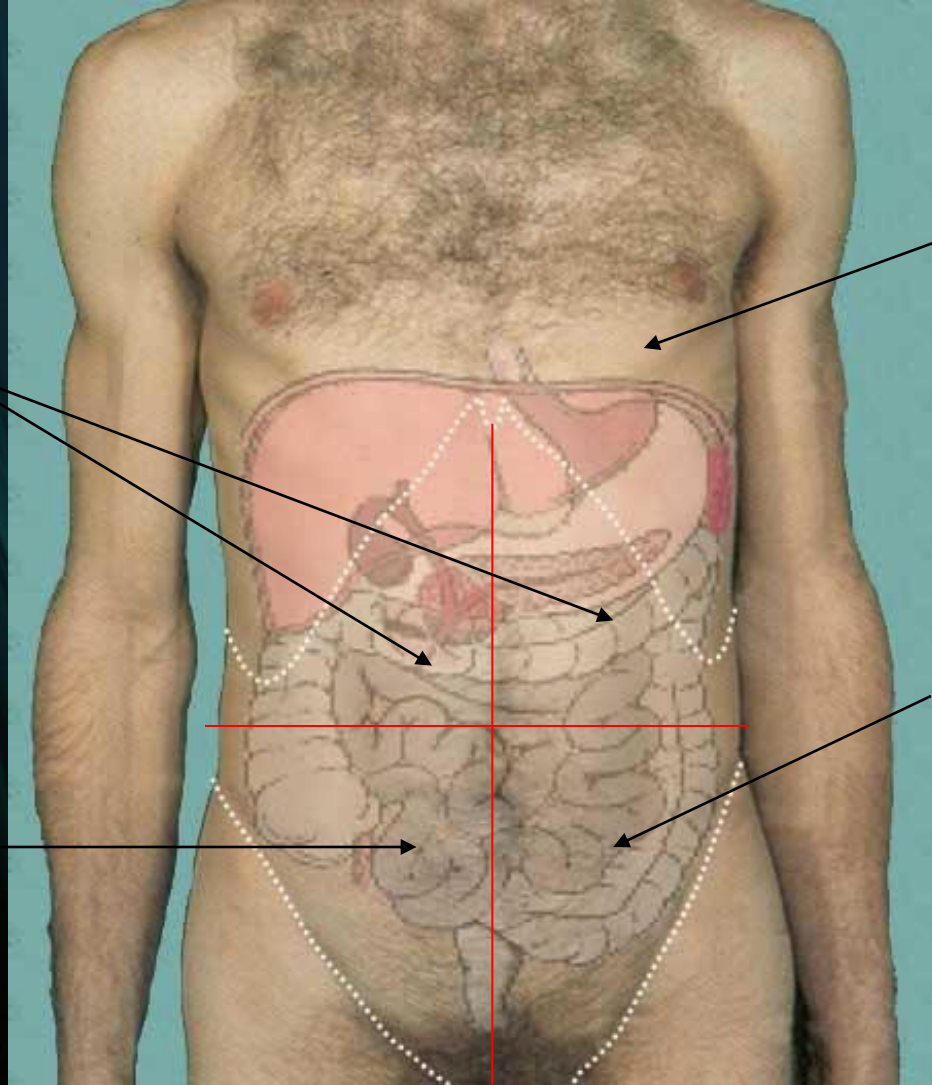
Not been clinically useful

- Cumbersome
- Require collection of a large amount of clinical and laboratory data over time
- Do not have acceptable positive and negative predictive value for severe acute pancreatitis

Bedside Index of Severity in Acute Pancreatitis (BISAP)

Risk Factors for Severity	Markers of Severity within 24 Hours
<ul style="list-style-type: none"> • Age >60 years • Obesity, BMI >30 • Comorbid disease <p style="text-align: center;">Markers of Severity during Hospitalization</p> <ul style="list-style-type: none"> • Persistent organ failure • Pancreatic necrosis • Hospital-acquired infection 	<ul style="list-style-type: none"> • SIRS [temperature >38° or <36°C (>100.4° or 96.8°F), Pulse >90, Tachypnea >24, WBC >12,000] • Hemoconcentration (Hct >44%) • <u>BISAP</u> <ul style="list-style-type: none"> • (B) Blood urea nitrogen (BUN) >22 mg% • (I) Impaired mental status • (S) SIRS: 2/4 present • (A) Age >60 years • (P) Pleural effusion • Organ Failure <ul style="list-style-type: none"> • Cardiovascular: systolic BP <90 mmHg, heartrate >130 • Pulmonary: Pao₂ <60 mmHg • Renal serum creatinine >2.0 mg%

Differential Diagnosis



- Pancreatitis
- Acute cholecystitis
- Ascending cholangitis
- Perforated viscus
- Appendicitis
- Caecal perforation
- Ruptured ectopic

- MI
- Severe pneumonia
- Intestinal obstruction
- Ruptured AAA
- Diverticulitis
- Bowel Ischaemia

Diagnosis

Requires two of the following:

- Typical abdominal pain
- $\geq 3X$ elevation in s. amylase and/or lipase level
- confirmatory findings on cross-sectional abdominal imaging

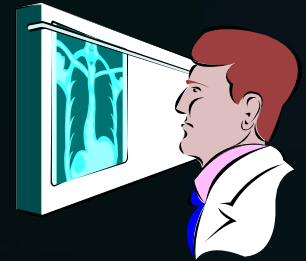
Diagnositics

- Elevated amylase, lipase, and urine amylase
- Elevated glucose, bilirubin, ALP, LDH
- Leucocytosis
- Hypocalcemia
- Hypomagnesia
- ST- T abnormalities
- **No** definite correlation between severity of pancreatitis and degree of lipase and amylase elevations
- After three to seven days, even with continuing pancreatitis, amylase values tend to return toward normal
- Lipase level is not elevated in diabetic ketoacidosis.



Diagnostics

- Abdominal USG
- CT, MRI, CXR
- ERCP
- EUS, MRCP



Diagnostics

CT

- Helpful in indicating the severity of acute pancreatitis, risk of morbidity and mortality , evaluating complications
- CT scan obtained within first several days of symptom onset may underestimate extent of injury
- Intestinal pancreatitis on initial CT scan may evolve to pancreatic necrosis on repeat CT scan 3-5 days later
- CT identification of local complications, particularly necrosis, is critical because patients with infected and sterile necrosis are at greatest risk of mortality

Complications (Systemic)

- Multisystem failure
- ARDS
- Coagulation defects– DIC
- Renal failure
- Fat necrosis
- Hypoglycemia
- Hypocalcemia
- Death (50 % of all deaths occur in first week)

Complications (Local)

- Necrosis
- Pancreatic fluid collections
 - Abscess
 - Pseudocyst
- Pancreatic ascites
- Involvement of contiguous organs
- Obstructive jaundice

Walled-off Necrosis

- Necrotizing pancreatitis - intense inflammatory response involving fat around the pancreas resulting in peripancreatic necrosis
- After three to six weeks, coalescence of pancreatic necrosis and peripancreatic fat necrosis into a structure encapsulated by fibrous tissue
- The walled-off necrosis contains semisolid necrotic tissue, liquefacted devitalized pancreatic and peripancreatic tissue
- Pancreatic necrosis does not become secondarily infected within 1st week of onset of acute pancreatitis

Pseudocysts

- Peripancreatic fluid collections persisting for > 4 weeks
- Pseudocyst should be distinguished from postnecrotic fluid collection
- Extrapancreatic collections of pancreatic fluid containing pancreatic enzymes and small amount of debris
- Walls consist of necrotic tissue, granulation tissue, fibrous tissue

Pseudocysts

- Elevated serum amylase
- Usually resolves spontaneously within few weeks
- Abdominal pain, with or without radiation to the back, is the usual presenting complaint
- Palpable, tender mass may be found in middle or left upper abdomen
- Rupture and hemorrhage are prime causes of death

Treatment

- In 85–90%, disease is self-limited and subsides spontaneously (3-7 days)
- **Analgesics** for pain
- **IV fluids** and colloids to maintain intravascular volume
- **No oral** alimentation
- **Nasojejunal enteral** nutrition considered over TPN since it maintains gut barrier integrity, less expensive, fewer complications
- When begin oral intake, should also be given pancreatic enzyme supplementation, PPI

Antibiotics

- Currently no role for prophylactic antibiotics in either interstitial or necrotizing pancreatitis
- Start antibiotics in patient who appears septic while awaiting the results of cultures. If cultures are negative, antibiotics should be discontinued
- Once a diagnosis of infected necrosis is established, appropriate antibiotics should be instituted and surgical debridement should be undertaken
- Sterile necrosis is most often managed conservatively

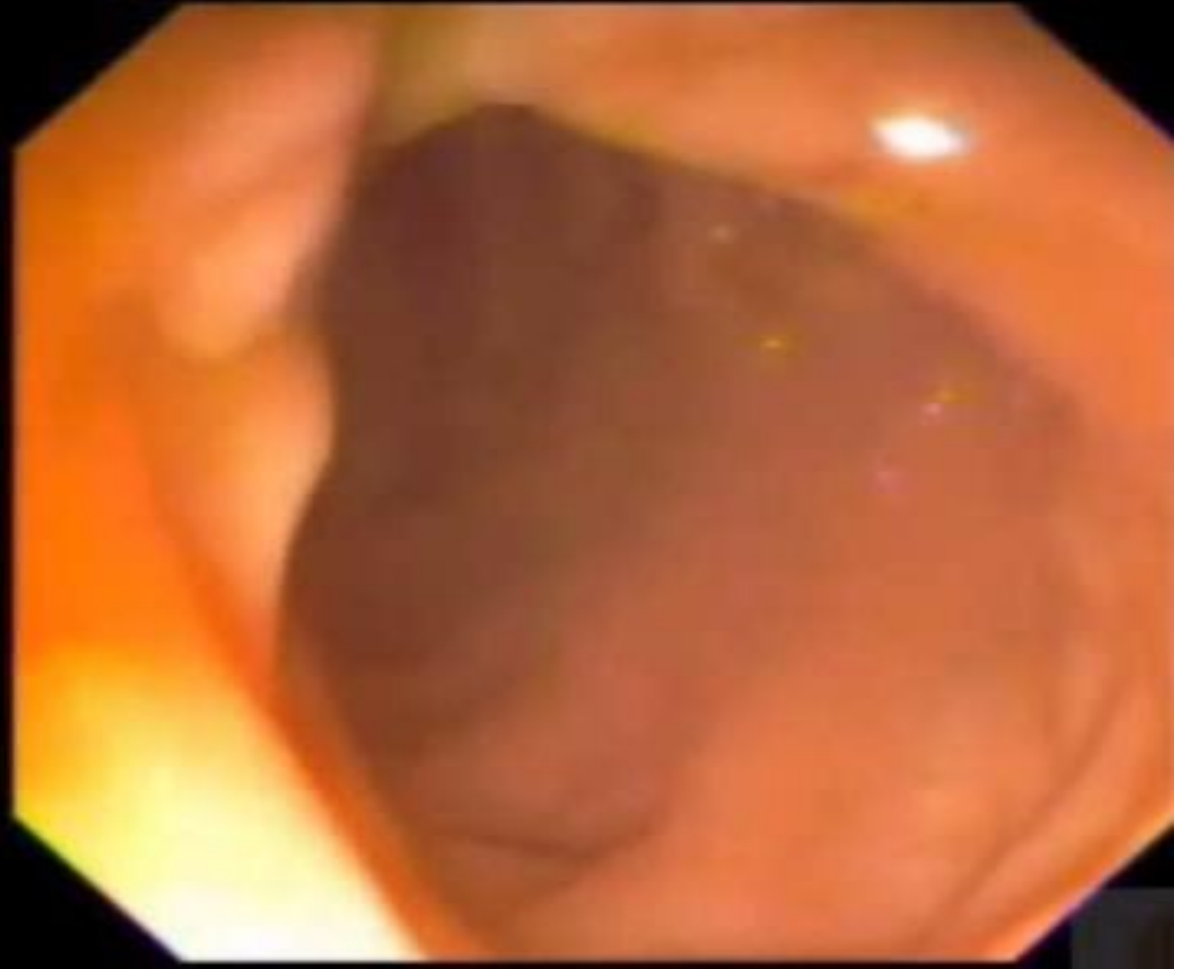
Treatment

- Urgent ERCP (24 hours) is indicated in severe acute biliary pancreatitis with organ failure and/or cholangitis
- Elective ERCP with sphincterotomy is indicated in persistent or incipient biliary obstruction, poor candidates for cholecystectomy, strong suspicion for bile duct stones after cholecystectomy
- ERCP with stent placement is also indicated for pancreatic ductal disruptions resulting in peripancreatic fluid collections

Endoscopic CBD stone removal

ID. No. :
Sex: Age:
D. O. Birth:
01/01/2000
00:10:54
CVP:
D. F:
Eth: L

Name :



Physician :

Treatment

- In patients who are stable and free of complications and serial USG show that the pseudocyst is shrinking, conservative therapy is indicated
- If pseudocyst is expanding and complicated by severe pain, hemorrhage, or abscess, patient should be operated
- Chronic pseudocysts can be treated safely and drainage can be accomplished by endoscopic or surgical means

Surgery – The Indications

1. For differential diagnosis
2. Persistent and severe biliary pancreatitis (not managed endoscopically)
3. Infected pancreatic necrosis
4. Drain pancreatic abscess (not managed percutaneously)

Indications that are less well defined / controversial

1. Presence of sterile pancreatic necrosis involving > 50% of pancreas
2. Persistent pancreatitis in spite of maximal medical therapy
3. When patient's condition deteriorates, often with the failure of one or more organ systems

Recurrent Pancreatitis

- 25% of patients have a recurrence
- Two most common factors are alcohol and cholelithiasis

In patients without obvious cause

- Occult biliary tract disease (microlithiasis) (66 %)
- Hypertriglyceridemia
- Drugs
- Pancreatic cancer
- Sphincter of Oddi dysfunction
- Pancreas divisum
- Cystic fibrosis

Chronic Pancreatitis

Irreversible damage to the pancreas

TIGAR-O Classification

<p>Toxic-metabolic Alcoholic Tobacco smoking Hypercalcemia Hyperlipidemia Chronic renal failure Medications—phenacetin Toxins—organotin compounds (e.g., DBTC)</p>	<p>Idiopathic Early onset Late onset Tropical</p>	<p>Genetic Cystic fibrosis Hereditary pancreatitis Cationic trypsinogen PRSS₁ PRSS₂ CFTR mutations SPINK1 mutations</p>
<p>Autoimmune Isolated autoimmune chronic pancreatitis Autoimmune chronic pancreatitis associated with Sjögren's syndrome Inflammatory bowel disease Primary biliary cirrhosis</p>	<p>Recurrent and Severe Acute Pancreatitis Postnecrotic (severe acute pancreatitis) Recurrent acute pancreatitis Vascular diseases/ischemia Postirradiation</p>	<p>Obstructive Pancreas divisum Sphincter of Oddi disorders Duct obstruction Preampullary duodenal wall cysts Posttraumatic pancreatic duct scars</p>

Clinical features

- Predominantly two symptoms:
 - abdominal pain or maldigestion
 - weight loss
- Abdominal pain may be quite variable in location, severity, frequency
- Eating may exacerbate the pain, leading to a fear of eating with consequent weight loss
- Maldigestion is manifested as chronic diarrhea, steatorrhea, weight loss
- Physical findings - usually unimpressive

Autoimmune Pancreatitis

- Mild abdominal pain, but without frequent pancreatitis
- Presentation with obstructive jaundice
- Diffuse swelling and enlargement of the pancreas, especially the head, mimicking carcinoma of pancreas
- Presence of other autoantibodies - ANA, RF
- Can occur with : Sjögren's syndrome, primary sclerosing cholangitis, UC, RA
- Absence of pancreatic calcifications or cysts
- Glucocorticoids are effective

Diagnostic Evaluation

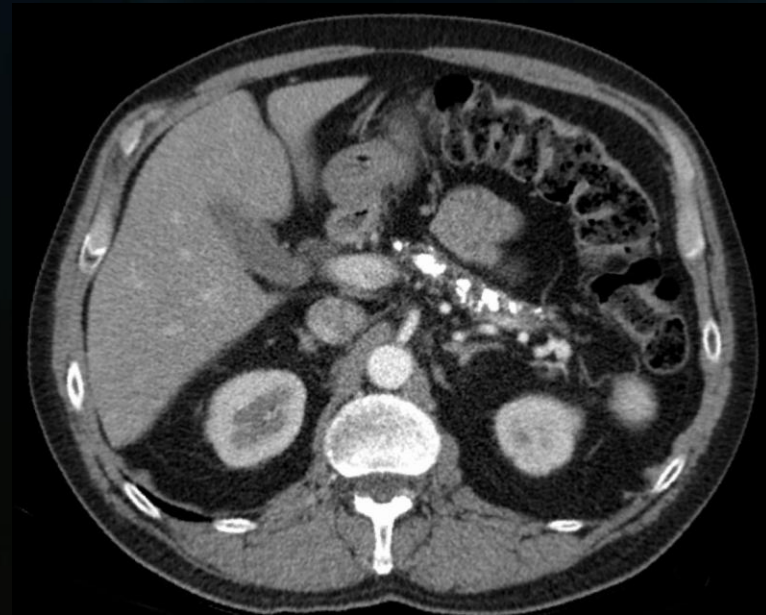
- Classic triad – pancreatic calcification, steatorrhea, DM (30%)
- Amylase & lipase are not ↑
- S. bil & S. alk phos may be ↑
- IGT & IFG may be present
- The diagnostic test with the best sensitivity and specificity is secretin
- It becomes abnormal when 60% of the pancreatic exocrine function has been lost

Tests of pancreatic exocrine function;

1. Secretin stimulation test
2. Study of intraluminal digestion products
3. Feecal pancreatic enzymes measurement eg. elastase

Diagnostic Evaluation

- Radiographic hallmark :
scattered calcification
throughout pancreas
- Abdominal USG, CT, MRCP
- MRCP provides a direct view
of the pancreatic duct and is
now the diagnostic
procedure of choice



Complications

- Narcotic addiction
- Impaired glucose tolerance
- Gastroparesis
- Cobalamin malabsorption
- Nondiabetic retinopathy
- Effusions with high amylase content GI bleeding
- Jaundice
- Cholangitis and/or biliary cirrhosis
- Subcutaneous fat necrosis
- Bone pain
- Pancreatic cancer

Treatment

- Two major issues – Pain Maldigestion

Pain –

1. Non narcotic analgesics
2. Avoid alcohol
3. Avoid large fatty meals
4. Endoscopic treatment involve sphincterotomy, stenting, stone extraction, and drainage of a pancreatic pseudocyst

Treatment

Maldigestion

- Pancreatic enzymes - cornerstone of therapy
- No consistent benefit to reducing pain
- Diarrhoea & steatorrhoea usually improved
- Prokinetic agents
- H₂ receptor blockers, PPI

Management - Surgical

- Chronic pancreatitis patients with dilated ducts and pain should be considered for surgical intervention
- Cholecystectomy - if biliary disease
- Pancreaticojejunostomy - pain relief
- Partial pancreatectomy - pain relief
- Vagotomy with gastric antrectomy - decrease enzyme production
- Whipple procedure as well as total pancreatectomy and autologous islet cell transplantation in selected patients with chronic pancreatitis and abdominal pain refractory to conventional therapy

Thank you



<http://dramiteshaggarwal.yolasite.com>

dramitesh@rediffmail.com