Gall bladder

Biliary ducts

extrahepatic

d. hepaticus  hepatic

d. cysticus  cystic

d. choledochus  common bile d.

intrahepatic

Pancreas
Diseases of the gall bladder

Gall stone disease
Cholecystitis
Tumors
Functional diseases
Gall stone D.

Location
Size
Infection
Gall stones in the gall bladder

Differences in size, shape, number, color, composition
Gall stones in gall bladder by US

Acoustic shadow
<table>
<thead>
<tr>
<th>Composition</th>
</tr>
</thead>
<tbody>
<tr>
<td>80% cholesterol</td>
</tr>
<tr>
<td>18% pigment – black st. -bilirubin brown stones (Asia) parasitic, intrahepatic</td>
</tr>
<tr>
<td>2% other -</td>
</tr>
</tbody>
</table>
Stone formation

Supersaturated bile

litogenic bile

BA – PhL – Chol imbalance

Nucleation

Hypomotility – DM2
Sludge $\rightarrow$ Stone

Mucin + others substances (cholesterol crystals, Ca-bilirubinat, Ca salts)
Clinical presentation

Silent stone
Biliary cholic
Cholecystitis
Jaundice- obstructive
Biliary pancreatitis
Biliary cholic

Pathomechanism – Anatomy - CCK

Onset
Character
Radiation
Accompanying symptoms
Diagnosis - Treatment
Epidemiology

Geographic differences — ethnicity, North Am.
At any age, but
The typical patient - 4F
Female
Forty y. o.
Fertile — pregnancy, oral contacept.
Fat - DM2
Native American Indian tribes

60%

African Masai tribe

0%
Further risk factors

Rapid weight loss
TPN (total parenteral nutrition)
Drugs - ceftriaxone, thiazide diuretics, octreotide, female sex hormones
Crohn disease -
Cirrhosis
Hemolysis
Reduced physical activity
Diet
Cholecystitis

In 90% gall stone associated

Inflammation - bacterial, *E. coli*, Klebsiella, Stre acute
chronic – repeated episodes

special form: Xanthogranulomatous (XGC)

Empyema

Gangrenous –

In 10% acalculous — in severely ill Pt., ICU Pt.
Murphy sign

Pain in midclavicular line at inspiration – terminated breath

Sonographic Murphy sign

95 % positivity in Acute cholecystitis
Diagnosis of Cholecystitis

Pain — Murphy sign
Fever
US
Blood tests —
  WBC, CRP
CT, HIDA (hepatic iminodiacetic acid)
Closed arrow points to gall bladder wall thickening. Open arrow points to stones in the GB.
Complications

Gangrene - fistula
Gallbladder rupture
Empyema
Fistula formation and gallstone ileus
Rokitansky-Aschoff sinuses

Warning signs:
  High fever, Shock
  Jaundice, Ileus

Carcinoma
Gall stone in the cystic duct

Symptoms of biliary cholic
Cholecystitis

Without jaundice

exception

Mirizzi syndrome

0.7-1.0 %

P.L.Mirizzi 1893-1964
Gall stone in the common bile duct

Leading symptom – obstructive jaundice
Risk of cholangitis – ascending infection
Risk of secondary biliary cirrhosis
Risk of biliary pancreatitis

Remove the stone!
Stone extraction by Dormia basket
Treatment of gall stone dis.

Spasmolytics
Pain killer
Antibiotics

Remove the gall bladder - >700,000 /year in USA
  Laparoscopic surgery
  Open surgery if necessary

Remove the stone - Endoscopic – ERPC
  Dormia basket
  Litotripsy
  Sphyncteterotomy (EST)
X-Ray during laparoscopic cholecystectomy
Pneumoperitoneum by Veress needle
Biliary tumors

Benign – papilloma, adenomyoma, cholesterol polyp

Malignant

Gall bladder carcinoma

Gallstone is a predisposing factor

Special form – Pocelain gallbladder

Intramural calcification

Prophylactic cholecystectomy
Biliary tumors 2.
Cancer of the biliary tree

Cholangiocarcinoma
Intrahepatic
Extrahepatic
periampullary
Klatskin tumor
hilar cholangiocarcinoma
Biliary or gallbladder dyskinezia

Motility disorder
Gall bladder and sphynctter Oddi dyskinezia
Biliary-type pain in absence of gallstone
  Intermittent - RUQ pain
  - nausea, vomiting

Spasm or scarring of SO
SO manometry

Treatment
  ERCP sphyncterotomy
  Cholecystectomy
Pancreatitis

Acute

Chronic

Recruent
ACUTE PANCREATITIS
Acute pancreatitis

Acute inflammation
Abdominal pain
Elevated pancreatic enzymes in serum
Self limiting
**INCIDENCE of ACUTE PANCREATITIS**

- **Varies in different countries**
- **Depends on cause**, e.g., alcohol, gallstones, metabolic factors, and drugs

**Estimated incidence**

- in England 5.4/100,000 per year
- in the USA 79.8/100,000 per year

185000 new cases of acute pancreatitis/y
Pathomechanism

**Autodigestion**

role of cytokines

Could be life-threatening

Acute abdominal catastrophe

However, majority is self limiting
Endocrine and Exocrine Pancreas Secretion

- Bicarbonate + 20 enzymes

- Insulin

- Glucagon
Classes of Enzymes in Pancreatic Juice

- Proteases: 90%
- Amylase: 7%
- Lipases: 2%
- Nucleases: <1%
Mechanism of Activation

- Zymogen
- Trypsinogen
- Protease
- Exposed catalytic domain
- TAP: Activation peptide
- Trypsin: Active enzyme
Why not everybody has pancreatitis?
Autodigestion is prevented by packaging of proteases in precursor form protease inhibitors. These protease inhibitors are found in the

- acinar cell
- pancreatic secretions
- $\alpha_1$- and $\alpha_2$-globulin fractions of plasma
Pain - abrupt onset, deep epigastric, often radiation to the back, belt form

History of previous episodes, often related to alcohol intake

Nausea, vomiting, sweating, weakness

Abdominal tenderness and distention

Fever

Lab. abnormalities
ACUTE PANCREATITIS

LABORATORY FINDINGS

Serum **amylase and lipase** are elevated within 24 hours in 90% of cases

**Leukocytosis** (WBC 10,000–30,000/µL)

**Proteinuria**

**Glycosuria** (10–20% of cases)

**Hyperglycemia** may be present
Elevated serum **bilirubin** may be present
Blood **urea nitrogen** and serum **ALP** may be elevated
Cytokine Production

Liver
- TNFα
- IL-1β
- IL-6

Lungs
- ICAM-1
- IL-1β
- TNFα
- PAF

Microcirculation
- PAF
- Endothelin
- INOS
- ICAM-1

Proinflammatory
Causes of pancreatitis

Common
Uncommon
Rare

Causes to consider in recurrent P. without any obvious etiology
Common Causes

Gallstones including microlithiasis
Alcohol acute and chronic alcoholism
Hypertriglyceridermia 
ERCP especially after biliary manometry
Trauma especially blunt abdominal trauma
Postoperative abdominal and nonabdominal op.
Drugs azathioprine, 6-mercaptopurine, sulfonamides, estrogens, tetracycline, valproic acid, anti-HIV medications
Sphincter of Oddi dysfunction
TRAUMATIC CAUSES

Blunt trauma  e.g. car accident
Penetrating trauma
Postoperative
ERCP  endoscopic retrograde cholangiopancreatography
Sphincter of Oddi manometry
Car accident is a risk for traumatic pancreatitis

Blunt trauma leads to injury of pancreas tissue
Acute pancreatitis
EMERGENCY SITUATION
Uncommon causes

Vascular causes and vasculitis
  ischemic-hypoperfusion states after cardiac surgery
Connective tissue disorders and
  thrombotic thrombocytopenic purpura (TTP)
Cancer of the pancreas
Hypercalcemia - hyperparathyroidism
Periampullary diverticulum
Pancreas divisum
Hereditary pancreatitis
Cystic fibrosis
Renal failure
Rare causes

Infections
mumps, coxsackievirus,
cytomegalovirus, echovirus, herpes V.
bacteria: tbc, leptospirosis
parasites ascaris, clonorchis

Scorpion toxin

Autoimmune
e.g., Sjögren's syndrome
Causes of Acute Pancreatitis (% of Cases)

- Gallstones: 45%
- Alcohol: 35%
- Other: 10%

Medications:
- Hypercalcemia
- Hypertriglyceridemia
- Obstructive
- Post-ERCP
- Hereditary
- Trauma
- Viral Vascular/ischemic
- Postcardiac bypass

**Idiopathic:** 10%
Gallstones

Common bile duct

Pancreas
OBSTRUCTIVE CAUSES

Gallstones
Ampullary/pancreatic cancer
Worms in pancreatic duct—ascaris
Choledochocele
Periampullary duodenal diverticula
Foreign body obstructing duct
Pancreas divisum with obstruction of accessory papilla
Hypertensive sphincter of Oddi
Acute Alcohol Effects

Abnormal blood flow and secretion

Sensitization to CCK

Zymogen activation

Cytokines

Stimulation of CCK and Secretine release

Sphyncter of Oddis spasm

Toxic metabolites
Oxidative
Non-oxidative
Pancreas is edematous and enlarged.
P. can show extensive necrosis, acute inflammation, suppuration and/or hemorrhage.
There can be extensive peripancreatic inflammation.
Fluid can accumulate in lesser sac and pleural space and paracolic gutters.
Neutrophils infiltrate the edge of the necrotic areas and extend into the adjacent lobules of fat and produce fat necrosis.
Necrosis of pancreatic parenchyma. In the center the parenchyma has been replaced by widespread autolysis and destruction by pancreatic proteolytic enzymes. Inflammatory cells.
Acute pancreatitis
Acute hemorrhagic pancreatitis
Pancreatitis - Steatonecrosis
Fat necrosis in acute pancreatitis
Severe Pancreatitis
Gray Turner sign
Emphysematous pancreatitis
Reduced proteases may increase CCK release
CCK Stimulates Pancreatic Enzyme Secretion by Neural and Hormonal Pathways
CCK hyperstimulation

Relevance to clinical pancreatitis

Organophosphorus insecticides  Scorpion toxins
Tityus trinitatis
PROGNOSIS of acute pancreatitis

Different scoring systems

Not US, but CT
Ranson's Criteria for predicting Mortality

**Present on Admission:**
- Age > 55 years
- WBC > 16,000/ul
- Blood glucose > 200 mg/dl
- Serum LDH > 350 I.U./L
- SGOT (AST) > 250 I.U./L

**Developing During the First 48 Hours:**
- Hematocrit fall > 10%
- BUN increase > 8 mg/dl
- Serum calcium < 8 mg/dl
- Arterial O2 saturation < 60 mm Hg
- Base deficit > 4 meq/L
- Estimated fluid sequestration greater than 6000 ml

Number of positive criteria
- 0-2 <5% mortality
- 3-4 20% mortality
- 5-6 40% mortality
- 7-8 100% mortality

N.B. serum amylase level is not one of criteria
"Acute Physiology And Chronic Health Evaluation" (APACHE II) score > 8 points predicts 11% to 18% mortality

Online calculator
Hemorrhagic peritoneal fluid
Obesity
Indicators of organ failure
Hypotension (SBP <90 mmHG) or tachycardia > 130 beat/min
PO2 <60 mmHg
Oliguria (<50 mL/h) or increasing BUN and creatinine
Serum calcium < 1.90 mmol/L (<8.0 mg/dL) or serum albumin <33 g/L (<3.2 g/dL)>
ACUTE PANCREATITIS – IMAGING

- US
- radiographs of the abdomen
  - gallstones
  - sentinel loop (segment of air-filled small intestine)
  - colon cutoff sign (gas-filled segment of transverse colon abruptly ending at the area of pancreatic inflammation)

- CT scan - gold standard!
A: Contrast-enhanced CT scan on admission of a patient with clinical evidence of acute pancreatitis.

B: Nine days after admission, there is a marked worsening with severe inflammation of the pancreas evidenced by anterior displacement of the posterior gastric wall.
CT scan in Acute Pancreatitis

Diffusely enlarged pancreas with low density from edema.

C: Colon
St: Stomach
P: Pancreas
CT scan in Acute Pancreatitis

- Phlegmon / Inflammatory mass
- White arrowheads: Phlegmon
- Black arrowhead: Pancreatic calcification
- Large Arrow: Peripancreatic fascial infiltration
- St: Stomach
## CT Severity Index (CTSI) in Acute Pancreatitis

<table>
<thead>
<tr>
<th>Grade of acute pancreatitis</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal pancreas</td>
<td>0</td>
</tr>
<tr>
<td>Pancreatic enlargement alone</td>
<td>1</td>
</tr>
<tr>
<td>Inflammation compared with pancreas and peripancreatic fat</td>
<td>2</td>
</tr>
<tr>
<td>One peripancreatic fluid collection</td>
<td>3</td>
</tr>
<tr>
<td>Two or more fluid collections</td>
<td>4</td>
</tr>
</tbody>
</table>

### Degree of pancreatic necrosis

<table>
<thead>
<tr>
<th>Degree of pancreatic necrosis</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>No necrosis</td>
<td>0</td>
</tr>
<tr>
<td>Necrosis of one-third of pancreas</td>
<td>2</td>
</tr>
<tr>
<td>Necrosis of one-half of pancreas</td>
<td>4</td>
</tr>
<tr>
<td>Necrosis of more than one-half of pancreas</td>
<td>6</td>
</tr>
</tbody>
</table>

**CT grade + necrosis score (0–10)**
Balthazar Scoring
for the Grading of Acute Pancreatitis

Grade A - normal CT
Grade B - focal or diffuse enlargement of the pancreas
Grade C - pancreatic gland abnormalities and peripancreatic inflammation
Grade D - fluid collection in a single location
Grade E - two or more collections and/or gas bubbles in or adjacent to pancreas
# ACUTE PANCREATITIS – SEVERITY INDEX WITH CT

<table>
<thead>
<tr>
<th>CT Grade</th>
<th>Points</th>
<th>Necrosis %</th>
<th>Additional Points</th>
<th>Severity Index</th>
<th>Mortality Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Normal pancreas</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>B Pancreatic enlargement</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>C Pancreatic inflammation and/or peripancreatic fat</td>
<td>2</td>
<td>&gt;30</td>
<td>2</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>D Single peripancreatic fluid collection</td>
<td>3</td>
<td>30-50</td>
<td>4</td>
<td>7</td>
<td>&gt;17</td>
</tr>
<tr>
<td>E Two or more fluid collections or retroperitoneal air</td>
<td>4</td>
<td>&gt;50</td>
<td>6</td>
<td>10</td>
<td>&gt;17</td>
</tr>
</tbody>
</table>
Complications of Acute Pancreatitis

Local
Systemic
Complications of Acute Pancreatitis

**LOCAL**

- Necrosis - sterile or infected
- Pancreatic fluid collections - abscess, pseudocyst
- Rupture
- Hemorrhage
- Infection
- Obstruction of gastrointestinal tract (stomach, duodenum, colon)
- Pancreatic ascites
- Disruption of main pancreatic duct
- Leaking pseudocyst
- Involvement of contiguous organs by necrotizing pancreatitis
  - Massive intraperitoneal hemorrhage
  - Thrombosis of blood vessels (splenic vein, portal vein)
- Bowel infarction
- Obstructive jaundice
Pancreatic pseudocyst

Cyst enucleated with a fistula communicating with distal pancreas

Transverse pancreaticojejunostomy

Postoperative wound infection
Complications of Acute Pancreatitis

SYSTEMIC

Pulmonary
Complications of Acute Pancreatitis

SYSTEMIC

Pulmonary

- Pleural effusion
- Atelectasis
- Mediastinal abscess
- Pneumonitis
- ARDS
Complications of Acute Pancreatitis

**SYSTEMIC**

Pulmonary
Cardiovascular

- Hypotension - shock
- Hypovolemia
- Sudden death
- Nonspecific ST-T changes in ECG simulating MI
- Pericardial effusion
Complications of Acute Pancreatitis

SYSTEMIC

Pulmonary
Cardiovascular
Hematologic

Disseminated intravascular coagulation (DIC)
Complications of Acute Pancreatitis

SYSTEMIC

Pulmonary
Cardiovascular
Hematologic
Gastrointestinal

GI bleeding
Peptic ulcer disease
Erosive gastritis
Hemorrhagic pancreatic necrosis with erosion into major blood vessels
Portal vein thrombosis,
Variceal hemorrhage
Ascites
Portal vein thrombosis in ac P

Longitudinal view of the portal vein in a patient shortly after acute pancreatitis, obstructive cholangitis.

Note the absence of CFM signals in the trombosed lumen (tr) and thickened wall of duodenum (d).
Portal vein thrombosis in ac P

The same view 16 months later shows partial recanalisation.

Resolution of clinical symptomatology with minimal ultrasound symptoms of portal hypertension - slightly enlarged spleen.
Complications of Acute Pancreatitis

**SYSTEMIC**

- Pulmonary
- Cardiovascular
- Hematologic
- Gastrointestinal

**Renal**

- Oliguria
- Azotemia
- Renal artery and/or renal vein thrombosis
- Acute tubular necrosis
Complications of Acute Pancreatitis

SYSTEMIC

Pulmonary
Cardiovascular
Hematologic
Gastrointestinal
Renal

Metabolic
  - Hyperglycemia
  - Hypertriglyceridemia
  - Hypocalcemia
Complications of Acute Pancreatitis

Systemic

Pulmonary
Cardiovascular
Hematologic
Gastrointestinal
Renal
Metabolic

Central nervous system
  Encephalopathy
  Sudden blindness (Purtscher's retinopathy)
Complications of Acute Pancreatitis

**SYSTEMIC**

Pulmonary
Cardiovascular
Hematologic
Gastrointestinal
Renal
Metabolic
Central nervous system

**Fat**

necrosis, emboli, subcutaneous tissue (erythematous nodules)
TREATMENT of acute pancreatitis
PRINCIPLES OF TREATMENT

- Intravascular volume - up to many litre
- Analgesia
- Put pancreas to ”rest” - somatostatin
- Nothing by mouth, nasogastric tube only for ileus or vomiting
- Treat complications — pulmonary, shock, renal, metabolic
- Remove obstructing gallstone in severe gallstone pancreatitis endoscopically
- Antibiotics for severe disease
- Percutaneous aspiration of pancreas to document infection in patient who fails to respond.
ACUTE PANCREATITIS – MINIMAL INVASIVE TREATMENT

• Gallstone in choledochus duct:
  – endoscopic papillotomy (EPT) + stone extraction

• Pseudocysts: endoscopic transpapillary or transgastric drainage
Endoscopic removal of pancreatitis causing gallstone
Enteral Feeding Sites

- Nasogastric
- Jejunal

To prevent CCK
ACUTE PANCREATITIS

ROLE OF SURGERY

• Débride necrosis
• Drain abscess/pseudocyst
• When diagnosis is in doubt
• Perforated viscus
• Small bowel obstruction/infarction
Scientists at the University of Liverpool have found how coffee can reduce the risk of alcohol-induced pancreatitis.
Approximately 25% of patients who have had an attack of acute pancreatitis have a recurrence.

The two most common etiologic factors:
- alcohol
- cholelithiasis

(others)
Relapsing pancreatitis often leads to chronic pancreatitis in which the pancreas has become so scarred that amylase and lipase levels no longer elevate in the blood.
CHRONIC PANCREATITIS
Chronic pancreatitis

- Pain
- Calcification
- Pancreatic insufficiency
- Progressive disease
PATHOPHYSIOLOGY OF CHRONIC PANCREATITIS

**Intraductal plugging and obstruction** - eg, ETOH abuse, stones, tumors

**Direct toxins** and toxic metabolites

**Release of cytokines** – also stimulate inflammation by neutrophils

**Stellate cell activation** to produce collagen and to establish **fibrosis**.

**Oxidative stress** - eg, idiopathic pancreatitis

**Necrosis-fibrosis** - recurrent acute pancreatitis that heals with fibrosis

**Ischemia** (from obstruction and fibrosis), important in exacerbating or perpetuating disease rather than in initiating disease

**Autoimmune disorders**

association with other autoimmune diseases, such as Sjögren syndrome, primary biliary cirrhosis, and renal tubular acidosis
Chronic pancreatitis
Chronic pancreatitis
Chronic pancreatitis
Fibrosing chronic pancreatitis
CHRONIC PANCREATITIS – ETIOLOGY

- Alcohol, 70%
- Idiopathic, 20%
- Other*, 10%
Alcoholic chronic pancreatitis

70%
Chronic pancreatitis other 10%

Pancreatic duct obstruction
  Tumor
  Trauma
  Pancreas divisum
  Fibrosis
Cystic fibrosis
Hyperlipidemia
Herediter pancreatitis
Tropical pancreatitis
Hyperparathyreoidism
CLASSIFICATION OF CHRONIC PANCREATITIS

- Calcifying
- Obstructive
- Inflammatory
- Autoimmune

Rome-Marseille, 1988
Autoimmune pancreatitis

Diagnostic criteria

P. imaging:
  diffuse narrowing of the main pancreatic duct with irregular wall (more than 1/3 of length of the entire pancreas).

Laboratory:
  IgG, (IgG4) ↑, Autoantibodies (ANA, anti-lactoferrin, RF)

Histology:
  Fibrotic changes with lymphocyte and plasma cell infiltrate

Responsiveness to corticosteroid treatment
Most common congenital anatomic variant of the human pancreas.

The embryologic ventral and dorsal pancreatic anlagen fail to fuse, so that pancreatic drainage is accomplished mainly through the accessory papilla.

The combination of pancreas divisum and a small accessory orifice could result in dorsal duct obstruction.

Endoscopic or surgical intervention is indicated only when the conservative methods fail.

If marked dilation of the dorsal duct can be demonstrated, surgical ductal decompression should be performed.

It should be stressed that the ERCP appearance of pancreas divisum—i.e., a small-caliber ventral duct with an arborizing pattern—may be mistaken as representing an obstructed main pancreatic duct secondary to a mass lesion.
SPHINCTEROTOMY OF THE MINOR PAPILLA
Hereditary pancreatitis

Mutation in cationic tripsinogen
Autosomal dominant
Incomplete penetrance
Early onset
Frequent calcification
Increased pancreatic cancer
Pancreatic Secretory Trypsin Inhibitor (PSTI) Gene Mutations

PSTI, or SPINK1, is a 56-amino-acid peptide that specifically inhibits trypsin by physically blocking its active site.
## CHRONIC PANCREATITIS

### DIAGNOSTIC TESTS

<table>
<thead>
<tr>
<th>FUNCTION</th>
<th>STRUCTURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct hormonal stimulation</td>
<td>ERCP</td>
</tr>
<tr>
<td>Bentriomin test</td>
<td>CT</td>
</tr>
<tr>
<td>Serum trypsin-like immunoreactivity</td>
<td>Endoscopis US</td>
</tr>
<tr>
<td>Fecal chymotripsin or elastase</td>
<td>MRI</td>
</tr>
<tr>
<td>Quantitative fecal fat</td>
<td>US</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>Plain abdomional radiograph</td>
</tr>
</tbody>
</table>

Tests are listed in order of decreasing sensitivity.
Chronic pancreatitis

Dilated main pancreatic duct and side branches.

Chronic stricture
Chronic pancreatitis

CALCIFICATION
Chronic pancreatitis

The Axial T2 Fat Saturation image on the right shows a dilated and irregular pancreatic duct.
Chronic pancreatitis - ERCP

dilated ducts, intraductal stones, strictures
Chronic pancreatitis and pseudocyst
The main PD is dilated and contains a calculus (arrow).

Note the acoustic shadow created by the stone.
Chronic pancreatitis

Irregular PD with hyperechoic margins and hyperechoic foci in the parenchyma.
Pancreatic pseudocyst

Large pseudocyst is visualised through the posterior gastric wall.

No mature wall is identified but the lesion is close to the gastric wall and no overlying vessels are seen.
Hyperamilasemia $\neq$ Pancreatitis
CHRONIC PANCREATITIS
COMPLICATIONS

- diabetes mellitus
- pancreatic pseudocyst or abscess
- cholestatic liver enzymes with or without jaundice
- common bile duct stricture
- malnutrition
- peptic ulcer
- pancreatic cancer develops in 4% of patients after 20 years
CHRONIC PANCREATITIS

PRINCIPLES OF TREATMENT

I.

• **Remove iniciting process**
  – Discontinue consumption of alcohol
  – Treat hyperlipidemia

• **Treat complications**
  – Pseudocyst
  – Duodenal obstruction
  – Common bile duct obstruction

• **Analgesics**
  – Non-narcotic
  – Narcotic
• Suppress pancreatic secretion
  – Pancreatic enzymes
  – Investigational agents

• Modify neural transmission
  – Celiac plexus block

• Relieve pancreatic ductal obstruction
  – Endoscopic surgery
  – Endoscopic stents (?)

• Remove pancreas through partial or complete surgery
Best in patients with recurrent acute pancreatitis caused by:
- cholelithiasis
- choledocholithiasis
- stenosis of the sphincter of Oddi
- Hyperparathyroidism

Patients not amenable to decompressive surgery, 
addiction to narcotics is a frequent outcome of treatment.
PRIMARY SCLEROSING CHOLANGITIS

PSC
ETIOLOGY / PATHOGENESIS

UNKNOWN

Genetic predisposition
Male: Female 2:1
Environmental factors?
„Disease of no-smokers”
Autoimmune mechanism – pANCA
Association with IBD
  UC – 50%
  Crohn – 2-4%
Predisposing for

Cholestasis
Bacterial cholangitis
Liver cirrhosis
Cholangio carcinoma — 3-36%
PSC histology
ANCA
Anti-neutrophil-granulocytica cytoplasmic antibody

cANCA

pANCA

Cytoplasmic binding

Perinuclear binding

In 80%
TREATMENT

Endoscopic dilatation
Antibiotics in bacterial infection
Liver transplantation
Chronic intrahepatic cholestasis – ALP, GGT ↑
Consequence of biliary epithelial cell injury
Female predominance (90%)
Autoimmune dis. – AMA M2 positivity
4 stages –
Treatment: Ursodeoxycholic acid, OLT

Will be discussed later