3rd Stage

hormones into blood vessels

Exocrine Pancreas

 About 85-90% of the pancreas is the exocrine pancreas that secretes enzymes important for the process of food digestion, 10-15% is the endocrine pancreas consist of islets of Langerhan's which secrete insulin, glucagon.



DISEASES OF EXOCRINE PANCREAS:

Acute pancreatitis

Inflammation of the pancreas associated with acinar cell injury, it is characterized by acute onset of abdominal pain resulting from enzymatic necrosis & inflammation of the pancreas. Typically pancreatic enzymes are elevated in serum & urine. The release of pancreatic lipases causes fat necrosis in and around the pancreas & with damage to the blood vessels causes acute hemorrhagic pancreatitis.

Morphology

4 main changes are associated with A.P:

- Proteolytic destruction of pancreatic substance .
- Necrosis of blood vessels with subsequent hemorrhage .
- Necrosis of fat by lipases .
- Acute inflammatory reactions .

MD5A





Acute pancreatitis

The most characteristic microscopic feature is the presence of fat necrosis which may spread throughout the abdominal cavity. These deposits may be evident as calcification on abdominal X-ray.

Pancreatic pseudocyst is a common complication of acute pancreatitis which does not contain epithelial lining.





Lecture	15	pathology
Lecture	10	pathology

3rd Stage

Pathogenesis

4 major etiologic factors are involved in A.P

<u>Metabolic</u>

Alcohol

Hypercalcemia

Drugs (e.g. thiazide diuretics)

<u>Mechanical</u>

Gall stones

Trauma

Surgery

<u>Vascular</u>

Shock

Atherosclerosis & embolism

Infectious

Mumps

The tissue lesions of acute pancreatitis suggest autodigestion of pancreas by activated pancreatic enzymes released from the cells with resultant proteolysis, lipolysis & weakening of blood vessels.

Activated trypsin convert other proenzymes to activate enzymes, prekallikrein to kallikrein activated kinin system & clotting system, leading to local inflammation, thrombosis & systemic clotting disturbances.

Mechanism for activation of pancreatic enzymes:

- 1. Cholelithiasis with impaction in ampulla of Vater result in obstruction & increased ductal pressure.
- 2. Alcohol :chronic alcohol ingestion causes secretion of protein rich pancreatic fluid which predispose to formation of calcified protein plugs.
- 3. Acinar cell injury: direct injury from alcohol, viruses, drugs, trauma & ischemia

Clinical features

A.P is a medical emergency acute abdomen, intense abdominal pain with upper back radiation, peripheral vascular collapse & shock.



Chronic pancreatitis

Termed chronic relapsing pancreatitis . Is characterized by repeated attacks of mild to moderate pancreatic inflammation with continuous loss of pancreatic panenchyma & replacement by fibrous tissue.

Most commonly affecting middle-aged men especially alcoholics. Biliary tract diseases play less important role in chronic pancreatitis than acute form, but hypercalcemia predispose to chronic pancreatitis. Many patients have no apparent predisposing factors.

Pathogenesis

Hypersecreation of protein from acinar cells with no excess fluid secretion causing precipitation of proteins with admixed with cellular debris to form *ductal plugs*, those plugs may enlarge in alcoholics to form *"stones*" containing ca- carbonate, end in calcification & ductal obstruction & atrophy of the draining pancreatic lobules.

Morphology

Irregularly distributed fibrosis, reduced number & size of acini with sparing of islets of Langerhan's with obstruction of pancreatic ducts, foci of calcification, stones may be present (*chronic calcifying pancreatitis*) in alcoholics.

Chronic obstructive pancreatitis with impacted stones around ducts. *Pseudocytes* formation is common clinically: silent, or recurrent attack of abdominal pain.

Late complications: diarrhea (malabsorption), steatorrhea, diabetes.

Pseudocytes:

Localized collection of fluid representing pancreatic secretion, almost always arising from bouts of acute or chronic pancreatitis not contain epithelial lining but fibrous inflammatory wall, usually unilocular.



3rd Stage

TUMORS

Benign tumors :

Benign tumors of the pancreas are rare

Cytadenomata are the main benign tumor of exocrine pancreas, they are painless, slowly growing tumor, multilocular, mucin-secreting (*benign mucinous cytadenoma*) or serous –secreting (*benign serous cyst adenoma*).



Pancr pseudocyst

Malignant tumors

Carcinoma of the exocrine pancreas, almost always arising from *ductal epithelial cells*.

Epidemiology

5% of all cancer death in U.S with an increasing incidence (due to smoking, diet, chemical carcinogens) occur in 6th -8th decade, smoker> nonsmoker, diabetics> non-diabetics.

Morphology

• Gross distribution head 60%, body 15%, tail 5% diffuse 20%. Maybe small or ill defined or large up to 10 cm with extensive local invasion & metastasis.





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Carcinoma of the head of the pancreas, the ampullary region is invaded & obstructing the bile outflow.Carcinoma of the body & tail not obstructs the biliary tract & remains silent



Microscopically

Adenocarcinomas arising from ductal epithilum, mucus secreting, many have abundant fibrous stroma. Most are moderately differentiated.

Clinically :

Pain in the epigastrium is usually the first symptoms.

Obstructive Jaurdice (carcinoma head of the pancreas)

Migratory thrombophlebitis.

Biochemical tests: CEA (carcinoembryonic antigen) & CA19-9 antigen are elevated in the serum but are not specific for carcinoma of pancreas.





Pancreatic carcinoma









Spread & metastasis :

Local causing obstructive Jaundice or invasion of duodenum,

Lymphatic, to adjacent lymph nodes

Hematogenous, to the liver

Prognosis

Prognosis of carcinoma of pancreas is extremely poor, only 10% of patients survive the first year after diagnosis.

Palliative surgery are performed to bypass the obstruction of the bile duct, relieving Jaundice & for obstruction of duodenum.

Islet cell tumor

- *A-Insulinoma (hyperinsulinism):*β-cell tumors are the most common of islet cell tumors, they are benign.
- Clinical triad :
 - > Attacks of hypoglycemia (blood glucose less than 50mg/dl)
 - > CNS manifestations (confusion ,stupor ,loss of consciousness).
 - Attacks are precipitated by fasting or exercise &relieved by feeding or i.v. glucose.

B-Gastrinoma (Zollinger-Ellison syndrome)

Hypersecretion of gastric acid with severe peptic ulceration in 90-95% of patients, duodenal, gastric & jejunal.

