Pancreatic diseases

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Anatomy



Retroperitoneal Organ Weighs 75 To 100 G 15 To 20 Cm Long Head Neck Body Tail

What is Pancreatitis?

Inflammation or infection of the pancreas

- Normally digestive enzymes secreted by the pancreas are not active until they reach the SI.
- When the pancreas is inflamed, the enzymes damages the tissue that produce them. attack and 2 types:

1. Acute Pancreatitis

2. Chronic Pancreatitis

Acute Pancreatitis

Definition and Incidence

- Inflammatory disease with little or no fibrosis.
- Initiated by several factors:
 - **90% of acute pancreatitis is secondary to acute cholelithiasis or ETOH** abuse
- Develop additional complications
- **300,000** cases occur in the united states each year leading to over 3000 deaths.

Etiology: (GET SMASHED)

- **G:** Gallstone
- E: Ethanol
- T: Trauma
- S: Steroid
- M: Mump
- A: Autoimmune
- S: Scorpion bits
- **H: Hyperlipidemia**
- E: ERCP
- **D:** Drugs

Clinical Presentation

Abdominal pain

- Epigastric
- Radiates to the back
- Worse in supine position
- Nausea and vomiting
- Garding
- Tachycardia, Tachypnea, Hypotension, Hyperthermia
- Elevated Hematocrit & Pre renal azotemia
- Cullen's sign
- Grey Turner's sign

Grey Turner sign



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Cullen's sign



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Diagnosis: Biochemical

<u>serum amylase</u>

- Nonspecific
- Returns to normal in 3-5 days
- Normal amylase does not exclude pancreatitis
- Level of elevation does not predict disease severity
- <u>Urinary amylase</u>
- <u>P-amylase</u>
- <u>Serum Lipase</u>

– <u>CBC</u>

- Increased Hb
- Thrombocytosis
- Leukocytosis

- Liver Function Test
 - Serum Bilirubin elevated
 - Alkaline Phosphatase elevated
 - Aspartate Aminotransferase elevated

Assessment of Severity

- Criteria
- 1.ranson
- 2.APACHE-2
- Biochemical Markers
- Computed Tomography Scan

Ranson Criteria Criteria for acute gallstone pancreatitis

- Admission
 - Age > 55
 - WBC > 16
 - Glucose > 200
 - LDH > 350
 - AST > 250

During first 48 hours

- Hematocrit drop > 10
- Pao2 less than 60mm Hg
- Serum calcium < 8</p>
- Base deficit > 4
- Increase in BUN > 5
- Fluid sequestration > 6l

CT scans of normal kidneys and pancreas



Pancreatic Necrosis



Treatmaent of Mild Pancreatitis

- Pancreatic rest
- Supportive care
 - fluid resuscitation watch BP and urine output
 - Pain Control
 - NG tubes and H₂ blockers or PPIs are usually not helpful
- Refeeding (usually 3 to 7 days) If:
 - Bowel Sounds Present
 - Patient Is Hungry
 - Nearly Pain-free (Off IV Narcotics)
 - Amylase & Lipase Not Very Useful

Treatment of Severe Pancreatitis

Pancreatic Rest & Supportive Care

- Fluid Resuscitation may require 5-10 liters/day
- Careful Pulmonary & Renal Monitoring ICU
- Maintain Hematocrit Of 26-30%
- Pain Control PCA pump
- Correct Electrolyte Derangements (K⁺, Ca⁺⁺, Mg⁺⁺)
- Contrasted CT scan at 48-72 hours
- Prophylactic antibiotics if present
- Nutritional support
 - May be NPO for weeks
- TPN

Complications

• Local

- Phlegmon, Abscess, Pseudocyst, Ascites
- Involvement of adjacent organs, with hemorrhage, thrombosis, bowel infarction, obstructive jaundice, fistula formation, or mechanical obstruction

• Systemic

- A. Pulmonary: pleural effusions, atelectasis, hypoxemia, ARDS.
- B. Cardiovascular: myocardial depression, hemorrhage, hypovolemia.
- C.Metabolic:Hypocalcemia,hyperglycemia,Hyperlipidemia,coagulopathy
- D. GI Hemorrhage
- E. Renal
- F. Hematologic
- G. CNS

H. Fat necrosis

Management



Chronic Pancreatitis

Definition and Prevalence

- Defined as chronic inflammatory condition that causes irreversible damage to pancreatic structure and function.
- Incurable
- 5 To 27 Persons Per 100,000





- Alcohol, 70%
- Idiopathic (including tropical), 20%
- Other, 10%
 - Hereditary
 - Hyperparathyroidism
 - Hypertriglyceridemia
 - Autoimmune pancreatitis
 - Obstruction
 - Trauma
 - Pancreas divisum

Classification:

- 1. calcific pancreatitis
- 2. obstruction pancreatitis
- **3. inflammatory pancreatitis**
- 4. auto immune pancreatitis
- **5. asymptomic fibrosis**
- 6. tropical pancreatitis
- 7. hereditary pancreatitis
- 8. idiopathic pancreatitis

Signs and Symptoms

- Steady And Boring Pain
- Not Colicky
- Nausea Or Vomiting
- Anorexia Is The Most Common
- Malabsorption And Weight Loss
- Diabetes

Laboratory Studies

Tests for Chronic Pancreatitis

- I. Measurement of pancreatic products in blood
 - A. Enzymes
 - **B.** Pancreatic polypeptide

II. Measurement of pancreatic exocrine secretion

- A. Direct measurements
 - 1. Enzymes
 - 2. Bicarbonate
- **B. Indirect measurement**
 - 1. Bentiromide test
 - 2. Schilling test
 - 3. Fecal fat, chymotrypsin, or elastase concentration
 - 4. [¹⁴C]-olein absorption

III. Imaging techniques

- A. Plain film radiography of abdomen
- B. Ultrasonography
- C. Computed tomography
- D. Endoscopic retrograde cholangiopancreatography
- E. Magnetic resonance cholangiopancreatography
- F. Endoscopic ultrasonography

Pancreatic calcifications. CT scan showing multiple, calcified, intraductal stones in a patient with hereditary chronic pancreatitis





Endoscopic retrograde cholangiopancreatography in chronic pancreatitis. The pancreatic duct and its side branches are irregularly dilated

features

CT

 The cardinal CT features of CP are pancreatic atrophy, calcifications, and main pancreatic duct dilation.



Treatment

- Analgesia
- Enzyme Therapy
- Antisecretory Therapy
- Neurolytic Therapy
- Endoscopic Management
- Surgical Therapy

Complications

- Pseudocyst
- Pancreatic Ascites
- Pancreatic-Enteric Fistula
- Head-of-Pancreas Mass
- Splenic and Portal Vein Thrombosis

Management



Annular Pancreas

- When the ventral pancreatic anlage fails to migrate correctly to make contact with the dorsal anlage, the result may be a ring of pancreatic tissue encircling the duodenum.
 - Such an annular pancreas may cause **intestinal obstruction** in the neonate or the adult.
- Symptoms of **postprandial fullness**, **epigastric pain**, **nausea**, and **vomiting** may be present for years before the diagnosis is entertained.



- The radiographic findings are symmetric dilation of the proximal duodenum with bulging of the recesses on either side of the annular band, effacement but not destruction of the duodenal mucosa, accentuation of the findings in the right anterior oblique position, and lack of change on repeated examinations.
- The differential diagnosis should include duodenal webs, tumors of the pancreas or duodenum, postbulbar peptic ulcer, regional enteritis, and adhesions.
 - Patients with annular pancreas have an increased incidence of pancreatitis and peptic ulcer.

Pancreas divisum

- Occurs when the **embryologic ventral and dorsal pancreatic anlagen fail to fuse**, so that pancreatic drainage is accomplished mainly through the accessory papilla.
- Is the **most common congenital anatomic variant** of the human pancreas.
- Does not predispose to the development of pancreatitis in the great majority of patients .
 - However, the combination of pancreas divisum and a small accessory orifice could result in dorsal duct obstruction.







MRCP of pancreas divisum



TUMOURS OF THE PANCREAS

The tumours of the pancreas can be -

- A. Non-Endocrine neoplasms
- **B. Endocrine neoplasms**

Malignant non-endocrine neoplasms. exocrine The most common are:-Ductal adenocarcinoma Cystadenocarcinoma

<u>NOTE</u>: Periampullary carcinoma is term used for juxtapancreatic carcinomas.

They are three forms:-

- Carcinoma of the ampulla
- Carcinoma of the lower CBD
- Duodenal carcinoma

ENDOCRINE NEOPLASMS:

These are less common than non-endocrine tumours and generally benign and sometimes multiple. They includes:

- Insulinoma
- Glucogonomas
- Others:
 - Gastrinomas
 - Somatostatatinomas
- Vipomas (Vasoactive Intestinal Polypeptide)

EVALUATION OF PANCREATIC NEOPLASMS:

- History
- Clinical Examination
- Investigations
- The specific investigations:-
 - Ultrasound Scan
 Histology & cytology
 - Angiography CT Scan
 - Laparoscopy MR Imaging
 - ERCP

NON-ENDOCRINE NEOPLASMS: (ADENO-CARCINOMA OF PANCREAS)

- Ductal adeno carcinoma (arising in the exocrine part of pancreas) account for 90% of pancreatic tumour 2/3rd located in the head of pancreas.
- Cystadenocarcinoma and endocrine tumour account for most of the remains of malignancy.

The exact causative factors responsible are unknown. The peak incidence in the 6th and 7th decade and more in men than women

The predisposing factors are:

- Diet (high protein & high fat)
- Smoking
- Exposure to industrial carcinogens

Spread of pancreatic tumours:

- A. Local Invasion
- **B.** Lymphatic
- C. Blood

causing D. Via peritoneal & omental ascites

CLINICAL FEATURES:

The diagnosis of pancreatic cancer varies from the simple and clinically obvious to the most difficult and almost impossible the initial symptoms and signs depend on the site and extent of the pancreatic cancer.

Modes of presentation:

- Weight loss
- Pain
- Jaundice
- Steatorrhoea
- Diabetes Mellitus
- Acute Pancreatitis
- Malignant Ascites
- Gastric Outlet Obstruction

- Approach to Investigations: (Selective Investigations)
- Ultrasound Scan
- C.T. Scan
- MR Imaging Scan
- ERCP
- Histology & Cytology
- Angiography (Coeliac, Superior Mesenteric)
- Laparoscopy

DELAY IN DIAGNOSIS:

Over 90% of patient with pancreatic cancer present in the late stage of their disease. At time no chance of cure.

The factors responsible for late diagnosis

stage. A. Tumour is asymptomatic in the early

- **B.** Patient delay.
- **C.** Physician delay.

D. The patient may not have ready and easy access to competent diagnostic center .

MANAGEMENT OF PANCREATIC CANCER:

A. Surgical Treatment

B. Non Surgical Treatment

SURGICAL TREATMENT:

Pancreatic Cancer is essentially incurable since metastasis occurs at such early stage. Any treatment must be regarded as palliative.

Surgical Options:

• For curative surgical treatment of cancer in the head of pancreas the optims are available:

A. Whipple operation (Pancreaticoduodenectomy)

B. Pylorus Preserving **Pancreaticoduodenectomy**

C. Total Pancreatectomy

 Palliative Surgical Treatment (Surgical Bypass)

For tail of the pancreas
 (Distal pancreatectomy)

Body of the pancreas
 the (Distal + removal of the body of pancreas)

for Pre-operative preparation of the patient major surgery:

- **good** 1. All jaundiced patients must be kept in state of nutrition and hydration.
- **corrected. 2.** Blood clotting deficiencies must be
- **3. Cardio pulmonary functioning carefully assessed.**
- cases. 4. Drainage procedure consider in certain

NON-SURGICAL TREATMENT:

The following options available: (Pallative procedure for non operable cases)

- Percutaneous coeliac ganglion blockade.
- (For pain)
- Stent to compress bile duct.

radiotherapy • Combination of chemotherapy and may become alterative in the future.

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<u>FUNCTIONING ENDOCRINE TUMOURS OF</u> <u>THE PANCREAS</u>:

These are much less common than adeno carcinoma.

The beta cell tumours secrete (Insulin) and called INSULINOMAS.

Another functioning tumour secrete(Gastrin) called GASTRINOMA which come from the islets which classified into either alpha cannot be or be (non-beta). Other tumours are:

Pancreatic a. Vipoma (Werner-Morrison syndrome, cholera)

b. Somastatinoma

c. Glucagonoma

Polypeptide tumours) d. HP (Human Pancreatic

Slow growing and therefore carry much better prognosis.

INSULINOMA:

The commonest islet cell tumour and arise from the beta cell and situated anywhere on the surface or within the substance of the pancreas.

 Most tumours are benign adenomas but 15% are low grade carcinomas and secrete (insulin). Whipple described a triad of features which typify the (insulinomas):

1. Fasting produces fainting.

2. During these "attacks" there is hypoglycaemia.

3. The attacks may be relieved by ingestion of glucose.

Overnight 1. Measurement of blood sugar in an attack. 2. insulin level (before & fasting serum glucose and after overnight). Insulin level are estimated by radioimmunoassay.

3.Pre-operative localization of the tumour very important identification at operation can be difficult.

[Combination CT Scan and selective angiography]

TREATMENT:

1.If the tumour localized surgical resection is the TR of choice also this apply to metastases.

2. If the tumours not localized during surgery (Intra operative USS can be done to localize the tumour) than resected.

3.Sub total distal resection for multiple tumours is appropriate.

4. With negative exploration it is appropriate to perform pancreatectomy distal to the superior mesenteric vessels.

5. The Hypoglycemic attacks may be relieved by diazoxide or streptazotocin.

<u>GASTRINOMA</u>: (Zollinger-Ellison Syndrome)

The tumour arising from the islets cell of langhans in the pancreas and in the duodenal wall.

The majority (60%) of these tumours are malignant. They may be associated with (MEN 1)which are Parathyroid Hyperplasia, and Pituitary Adenoma. Gastrinoma give rise to ZE Syndrome which consist of triad (hypersecretion of gastric acid, severe peptic ulceration and the presence of non-beta cell tumour of the pancreas or duodenum). The disease present as peptic ulcer disease in 90%.They have typical pain more over severe and less response to medical treatment.

Co-existing diarrhoea.

All complications of peptic ulcer disease are present in (ZE-Syndrome) as acute haemorrhage, perforation and recurrent ulceration.

THE DIAGNOSIS OF ZE-SYNDROME:

•Severe peptic ulcer disease doesn't respond to treatment.

•Multiple peptic ulcers or ulcers in unusual distal duodenum or locations such as the jejunum.

Peptic ulcer disease associated with diarrhoea.

 Recurrent peptic ulcer disease following in acid reducing operation (surgery).

Peptic ulcer is associated with MEN- 1 Syndrome.

Marked elevation of serum gastrin.

TREATMENT:

Medical therapy for control of the acid hypersecretion in patient with ZE-Omprazole considered the Syndrome antisecretory drug of choice for all gastrinoma patients.

- Surgical Treatment:
- **Tumour excision.**
- **Total gastrectomy.**

Patient with metastases should have medical therapy if fail total gastrectomy.

Gastrinoma patient with MEN 1 Syndromeand hyperparathyroidism should have documented parathyroid surgery performed prior to removal of gastrinoma.

