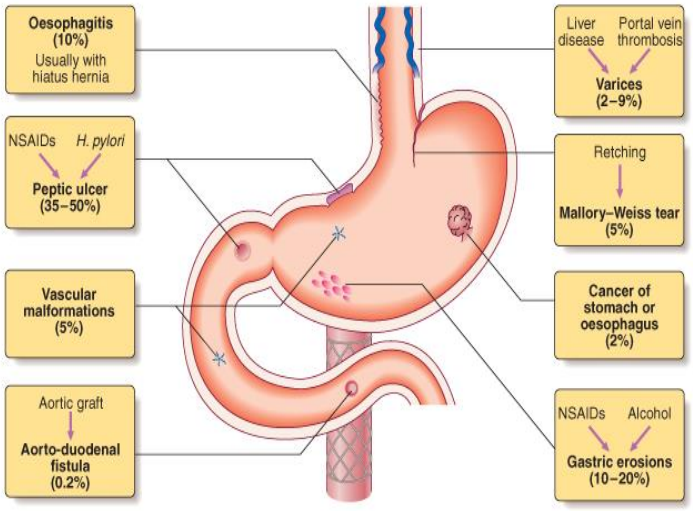
Lecture 8[2hrs]

Gastro-Intestinal Bleeding

Bleeding from the gastrointestinal (GI) tract may present in 5 ways.
1-Haematemesis [vomitus of red blood or "coffee-grounds" material] commonly related to bleeding from the esophagus, stomach, and duodenum.
2-Melena is black, tarry, foul-smelling stool. Occur when as little as 50-100 ML of blood enter the GIT tract [can originate from the esophagus, stomach, small bowel, and proximal colon]
3-Hematochezia is the passage of bright red or maroon blood from the rectum.
4-Occult GI bleeding, subacute bleeding that is not clinically visible.
5-Obscure GIB
acute or chronic bleeding for which source of bleeding cannot be identified after routine endoscopic evaluation with OGD, colonoscopy including small bowel.
GIT bleeding may be upper or lower in origin.
Causes of GIT bleeding include
1-Causes of upper GIT bleeding [intraluminal blood loss proximal to the ligament of treitz]



*Vascular malformations [including hereditary hemorrhagic telangiectasia's (Osler-Weber-Rendu) and gastric antral vascular ectasia ("watermelon stomach") ,[Dieulafoy's lesion [in which an aberrant vessel in the mucosa bleeds from a pinpoint mucosal defect].
*Prolapse gastropathy (Prolapse of the proximal stomach into the esophagus with retching especially in alcoholics)
*Hemobilia [bleeding from the bile duct] and Hemosuccus pancreaticus [bleeding from pancreatic duct] **Mucosal Tear (Mallory-Weiss Syndrome)**
The tear usually involves the gastric mucosa near the squamocolumnar junction.
Usually caused by vomiting, retching, or vigorous coughing. Patients present with upper gastrointestinal bleeding, which may be severe. In most patients bleeding stop spontaneously; continued bleeding may need vasopressin therapy, angiographic embolization or Surgery
2-Lower GIT bleeding. [bleeding distal to the ligament of treitz]
Causes include
***Small intestinal**
Bleeding from sites beyond the reach of the standard upper endoscope is difficult to diagnose and is responsible for the majority of cases of obscure GIB.
The most common causes are
*Vascular ectasia and tumors (e.g., adenocarcinoma, leiomyoma, lymphoma, benign polyps, Carcinoid, metastases, and lipoma).
*Less common causes include Crohn's disease, infection, ischemia, vasculitis, small bowel varices, diverticula, Meckel's diverticula, duplication cysts, and intussusceptions.

*NSAIDs induce small intestinal erosions and ulcers and may cause chronic obscure GIB.
*Meckel's diverticulum is the most common cause of significant LGIB in children.

Colonic

*Hemorrhoids are the most common cause of LGIB
*Anal fissures.
*Diverticula, vascular ectasias (especially in the proximal colon of patients > 70 years)
*Neoplasm's (adenomatous polyps and adenocarcinoma),
*Colitis most commonly infectious or idiopathic inflammatory bowel disease
*Ischemic colitis or radiation-induced.

Uncommon Causes of LGIB include

*Post-polypectomy bleeding. *Solitary rectal ulcer syndrome.
*Ectopic varices (most commonly rectal), lymphoid nodular hyperplasia, vasculitis, and aorto-colic fistulas. In children and adolescents, the most common colonic causes of significant GIB are inflammatory bowel disease and juvenile polyps

Differentiation of Upper from Lower GIB.

*A non-bloody NG aspirate doesn't exclude UGIB.
*Hematemesis indicates an upper GI source of bleeding (above the ligament of Treitz).
*Melena indicates that blood remains in the GI tract for at least 14 h. Thus the more proximal the bleeding site the more likely Melena will occur.
*Hematochezia usually represents a lower GI source of bleeding, although an upper GI lesion may bleed so rapidly that blood does not remain in the bowel long enough for melena to develop. When hematochezia is the presenting symptom of UGIB it is associated with hemodynamic instability and dropping hemoglobin.
*Bleeding lesions of the small bowel may present as melena or hematochezia.
*Other clues to UGIB include hyperactive bowel sounds and an elevated BUN [Due to volume depletion and absorbed blood proteins.]

Investigations and management of patients with GIT Bleeding include.

Clinical Assessment

*Measurement of the heart rate and blood pressure is the best way to assess a patient with GIB. Clinically significant bleeding leads to postural changes in heart rate or blood pressure, tachycardia, and recumbent hypotension[postural hypotension of 10 mmHg or higher coupled with arise in pulse rate of greater than 20 beats per minute indicate at least a 20% reduction in blood volume]. Recumbent systolic blood pressure below 100 mmHg or pulse rate above 110 beats per minute usually indicates that more than 40% of the blood volume has been lost.
*Assess Rockall score to predict mortality risk following upper GIT bleeding

Criteria score	o----------	score1------	--score 2----------	score 3---------
Age	<59	60-79		>80
shock	no shock	Pulse > 100		BP<100 mmHg
Comorbidity	none		CHF, IHD	RF, liver, metastatic cancer
Source of bleeding	Mallory-Weiss	PUD,varices		malignancy
Stigmata of recent bleeding	non-adherent clot		Spurting vessel	

Rockall score >3 good prognosis >8 high risk

1-Intravenous access. At least one large-bore cannula.

2-Resuscitation.

*Intravenous crystalloid fluids or colloids are given to restore the blood pressure.
*Blood is transfused when the patient is shocked, Hb less than 10gr%.
*Normal saline should be avoided in patients with liver disease because it can worsen the Ascites.
*CVP [central venous pressure] is used in severe bleeding particularly in a patient with cardiac disease.

3-Oxygen. By face mask to all shocked patients.

4-Blood tests.

*The hemoglobin does not fall immediately with acute GIB, due to proportionate reductions in plasma and red cell volumes. Thus, hemoglobin may be normal or only minimally decreased at the initial presentation of a severe bleeding episode. As extravascular fluid enters the vascular space to restore volume the hemoglobin falls but this process may take up to 72 h. Patients with slow chronic GIB may have very low hemoglobin values despite normal blood pressure and heart rate
*Urea and electrolytes.
*Liver function tests.
*Prothrombin time if there is a clinical suggestion of liver disease or ant coagulated patient.
*Cross-matching of at least 2 units of blood.
5-Monitoring. Hourly *blood pressure*Pulse rate*urine output.
6-Endoscopy *OGD for UGIT Bleeding after resuscitation to establish the site of bleeding and therapy**.**
***Colonoscopy** to exclude colonic cause of LGITB after resuscitation to establish the site of bleeding and therapy**.**
If both OGD and colonoscopy are normal and site of bleeding cannot be identified then
a-Enteroscopy, single and double Balloon Enteroscopy, intraoperative endoscopy
b-wireless capsule endoscopy
c-Technetium labeled nuclear scan. Requires a flow rate of only 0.1ml/mint
may show bleeding from Meckel's diverticulum
d-Visceral angiography is indicated when Endoscopy is normal and the patient is actively bleeding by at least 1ml per minute to allow for immediate therapy with arterial embolization
7-Surgical operations indicated when
*Failure of Endoscopical therapy.
*Rebreeding occurs on one occasion in an elderly or frail patient or twice in younger.
patients following successful therapy of ulcer bleeding
* H. Pylori should be eradicated*patients should avoid NSAIDs.

DISEASES OF THE PANCREAS

Pancreatic Secretion includes
*Endocrine hormones, insulin, and glucagon, controlling glucose homeostasis.
*Exocrine pancreatic secretions.
*Pancreatic digestive enzymes are secreted by pancreatic acinus and sodium bicarbonate solution are secreted by ducts. Both products flow through the pancreatic duct that joins the common bile duct before it empties into the duodenum through the papilla of Vater.
Pancreatic juice is secreted in response to the presence of chyme in the upper portions of the small intestine. When synthesized in the pancreatic cells the proteolytic digestive enzymes are in the inactive forms. They become activated only after they are secreted into the intestinal tract. Trypsinogen is activated to trypsin by an enzyme called enterokinase which is secreted by the intestinal mucosa. bicarbonate ions play an important role in neutralizing the acidity of the chyme emptied from the stomach into the duodenum. The proteolytic enzymes of the pancreatic juice must be activated only after reaching the intestinal lumen otherwise if activated inside the pancreas it will digest the pancreas itself causing acute pancreatitis within a few hours. The same cells that secrete proteolytic enzymes secrete at the same time trypsin inhibitor. [This substance prevents activation of trypsin both inside the secretory cells and in the acinus and ducts of the pancreas].
Pancreatic secretion contains multiple enzymes for digesting all of the three major types of food: proteins, carbohydrates, and fats.
*The Pancreatic enzyme for digesting proteins is trypsin, chymotrypsin, and carboxypolypeptidase. Trypsin and chymotrypsin digested proteins into peptides of various sizes.
*The pancreatic enzyme for digesting carbohydrates is pancreatic amylase, which hydrolyzes starches and glycogen to form disaccharides. which can then be cleaved to glucose by brush border enzymes in the small intestinal mucosa

*The main enzymes for fat digestion are
1- pancreatic lipase which is capable of hydrolyzing neutral fat into fatty acids and monoglyceride.
2- cholesterol esterase which causes hydrolysis of cholesterol esters
3- phospholipase, which splits fatty acids from phospholipids.

Regulation of Pancreatic Secretion

Stimuli for the regulation of pancreatic secretion include
1. **Acetylcholine.** which is released from the parasympathetic vagus nerve endings and cholinergic nerves in the enteric nervous system. stimulate the acinar cells of the pancreas causing the production of large quantities of pancreatic digestive enzymes.
2. **Cholecystokinin.** which is secreted by the duodenal and upper jejunal mucosa when food enters the small intestine. CCK stimulates the acinar cells of the pancreas causing the production of large quantities of pancreatic digestive enzymes.
3. **Secretin**

Secreted by the duodenal and jejunal mucosa when acidic food enters the small intestine. secretin Stimulates secretion of large quantities of water solution of sodium bicarbonate by the pancreatic ductal epithelium.

Acute Pancreatitis

Acute pancreatitis is an inflammatory process that occurs on the background of a previously normal pancreas and can return to normal after the resolution of the episode. characterized by severe abdominal pain and loco-regional and systemic inflammatory complications. acute pancreatitis maybe
* Edematous pancreatitis - Characterized by interstitial inflammation and edema with peri-pancreatic fat necrosis but sparing acinar cells.
*Necrotizing pancreatitis in which acinar cells are destroyed.
*Acute hemorrhagic pancreatitis which is characterized by bleeding into the pancreas and retro-peritoneum

Causes of acute pancreatitis include

Gall stones, Alcohol, Idiopathic, Post-ERCP, Post-surgical, Trauma,
Drugs [azathioprine, thiazide, Sodium valproat]
Metabolic [hypercalcimia,hypertriglyceridimia]
Pancreas divisum.results when the ducts of embryologic ventral and dorsal parts of the pancreas fail to fuse.wirsung s duct which normally drains the entire pancreas only drain the uncinate process in these patients. The rest of the pancreas is drained by the duct of Santorini through the minor papilla.
Infection [mumps,coxsackie virus]
Hereditary pancreatitis is autosomal dominant disease symptoms appear between the age of 5—15 years and progress to chronic pancreatitis.
Renal failure
Organ transplantation
Penetrating duodenal ulcer

PATHOGENESIS

Acute pancreatitis is a consequence of premature activation of pancreatic enzymes in the pancreas rather than in the intestinal lumen. The activated enzymes lead to autodigestion and cellular injury vascular damage, coagulation, fat necrosis, and parenchymal cell necrosis and vasoactive substances (histamine, bradykinin) that are released these vasoactive substances causes [Vasodilatation, Increased vascular permeability, edema, and inflammation].

Factors that may initiate the activation of the pancreatic enzyme include

1-Reflux of infected bile or duodenal contents into the pancreatic duct
2-Defective intracellular transport and secretion of pancreatic zymogens
3-Hyperstimulation of the pancreas by alcohol or fat.
4-Pancreatic duct obstruction by Choledocholithiasis or tumors.

The severity of acute pancreatitis is dependent upon the balance between the activity of released proteolytic enzymes and anti-proteolytic factors that include [Intracellular pancreatic trypsin inhibitor protein and circulating alpha 2-macroglobulin, alpha 1- antitrypsin and C1esterase inhibitors]. The normal pancreas has only poorly developed capsules and adjacent structures including CBD, duodenum, splenic vein, and transverse colon is commonly involved in the inflammatory process**.**

Clinical Features include

***Abdominal pain**

The pain usually begins in the epigastrium and radiates to the back, chest, flanks, and lower abdomen.

The pain is more intense when the patient is supine and the patient obtains relief by sitting with the trunk flexed and knees were drawn up.

*Nausea, vomiting, and abdominal distention [due to gastric, intestinal hypomotility and chemical peritonitis].

Physical examination

*Distressed and anxious patient. *Low-grade fever, tachycardia, and hypotension.

*****Jaundice **[**Due to edema of the head of the pancreas with compression of the intra-pancreatic portion of the common bile duct].

*****Erythematous skin nodules due to subcutaneous fat necrosis.

*****Pulmonary findings, including basilar crepitations, atelectasis, and left-sided pleural effusion.

*****Abdominal tenderness and muscle rigidity.

*****Bowel sounds are usually diminished or absent.

Signs of hemorrhagic pancreatitis include

*****Cullen's sign [blue discoloration around the umbilicus]**.**

*****Turner's sign [a green-brown discoloration of the flanks]. Both suggest hem peritoneum

Adverse prognostic factor in acute pancreatitis

The Ranson criteria consist of 11 clinical signs with prognostic significance 5 criteria are measured at admission and 6 are measured between admission and 48 hrs the number of Ranson criteria correlates with the incidence of complications.

Ranson criteria for assessing the severity of acute pancreatitis

Measured at time of admission

Age >55years

Leukocytosis >16000

Glucose >200mg/Dl

LDH> 350U/L

Aspartate aminotransferase > 250 u/L

Measured during the initial 48 hrs

Hematocrit decreases >10%

PaO2 <60 mm Hg

Ca < 8mg /dL

Urea nitrogen increases >5 mg/dL

Base deficit >4 mEq/L

Fluid sequestration >6 L

Severity and prognosis worsen as the number of these factors increases. Patients with Ranson criteria more than or equal to 3 should be admitted to ICU and may need surgical intervention.

The differential diagnosis of acute pancreatitis includes. **1-**perforated viscus. **2-** Acute cholecystitis and biliary colic.**3-** acute intestinal obstruction.**4-** mesenteric vascular occlusion.

5- Renal colic.

6- Myocardial infarction.**7-** dissecting aortic aneurysm. **8-** Connective tissue disorders with vasculitis.

9- Pneumonia.**10-** diabetic ketoacidosis.

Complications of acute pancreatitis include.
The retroperitoneal location of the pancreas and the absence of a well-developed capsule allow the inflammatory process to spread freely. The activated pancreatic enzymes dissect through the tissue planes and affect the upper abdominal organs and peritoneal surfaces may be involved with the inflammatory process leading to exudation and fluid accumulation in the peritoneal cavity. involvement of diaphragmatic lymphatics may lead to pleural effusion and pneumonitis. Pancreatic enzymes and vasoactive substances can cause leakage of protein-rich fluid from the systemic circulation into peritoneal and retroperitoneal spaces causing hypovolemia.

1-Systemic complications include

*Systemic inflammatory response syndrome [SIRS] and renal failure.
*****Paralytic ileus and vomiting.
*Hypoxia due to acute respiratory distress syndrome [non-cardiogenic pulmonary edema due to disruption of the alveolar-capillary membrane causing filling of alveolar space by a transudate],
*Fat necrosis.
*Disseminated intravascular coagulation and microthrombi due to the release of activated pancreatic enzymes into the circulation.
*Circulatory shock.

2-Pancreatic complications

*Pancreatic Abscess [circumscribed collection of pus close to the pancreas and containing little or no pancreatic tissue]
*Pseudocyst. [Are collections of tissue, fluid, debris, pancreatic enzymes, and blood which develop throughout 1 to 4 weeks after the onset of acute pancreatitis due to Disruption of pancreatic ducts. In contrast to true cysts, pseudocysts do not have an epithelial lining, majority pseudocyst are located in the body or tail of the pancreas. cyst May be single or multiple
*Pancreatic Ascites

3-Gastrointestinal complications

*Bleeding from -Gastric, duodenal or colonic erosion.
-Variceal hemorrhages [due to splenic or portal vein thrombosis].
*Duodenal obstruction [caused by compression of the duodenum by pancreatic mass]
*Obstructive jaundice [compression of pancreatic duct by pancreatic mass].

1nvestigations.

***Increased level of serum amylase**. But After 48 to 72 h, even with continuing evidence of pancreatitis, serum amylase returns to normal. And in this situation, the diagnosis can be made by demonstrating an elevated urinary amylase/ creatinine ratio.
***Serum Lipase** levels may remain elevated for 7 to 14 days.
*Leukocytosis
*Hyperglycemia
*Hypocalcaemia due to sequestration of calcium in fatty necrosis
*Serum bilirubin, alkaline phosphatase, and ALT may be elevated
*Arterial hypoxemia
***Plane abdominal X-rays** to exclude a perforated viscus and to see the colon cutoff sign [refers to the abrupt narrowing of the gas in the transverse colon nearby the body of the pancreas].
***US.** to assess pancreatic size which may be normal in the early stage and to evaluate the gallbladder and biliary tree.
***Abdominal CT** scans in the early stage shows pancreatic swelling and between 3-10 days to define the viability of the pancreas. In necrotizing pancreatitis is associated with decrease enhancement following intravenous injection of contrast material, presence of gas within necrotic material suggests infection and abscess formation which needs aspiration for bacterial culture. CT scan also shows the involvement of colon, blood vessels by the inflammatory process.

TREATMENT

The disease is self-limited in approximately 85 to 90% of patients and resolves spontaneously usually within 3 to 7 days after treatment.

* Severe cases should be managed in the intensive care unit [Ranson criteria >3].

*A central venous line, a urinary catheter is used to monitor patients with shock.

*****Analgesics like pethidine.

*****Intravenous fluids and colloids to maintain normal intravascular volume.

*****Feeding by Naso- enteral tube should be started at an early stage in patients with severe pancreatitis.

*****Nasogastric suction is indicated only when paralytic ileus is present**.**

*****Oxygen for a hypoxic patient.

*****Ventilatory support [for a patient with ARDS]

*****Insulin for hyperglycemia.

*****Intravenous calcium if Tetany occurs.

*****Prophylaxis for thromboembolism with subcutaneous heparin.

*Prophylactic broad-spectrum intravenous antibiotics may improve outcome in severe cases.

*****ERCP for a patient with cholangitis or jaundice.

*****Surgery for necrotizing pancreatitis or pancreatic abscess.

*****Distal pancreatectomy for pancreatic Ascites.

Chronic Pancreatitis

Chronic pancreatitis is a chronic inflammatory disease characterized by fibrosis and destruction of exocrine pancreatic tissue. Diabetes mellitus occurs in advance cases because the islet of Langerhans is involved.

Causes of chronic pancreatitis.

1-Calcific chronic pancreatitis includes [alcoholism, tropical]. 2-Obstructive chronic pancreatitis causes include [stenosis of the ampulla of Vater]. 3-Cystic fibrosis.

4- Idiopathic chronic pancreatitis.5- Hereditary.6-pancreas divisum

7-Metabolic causes include- hypertriglyceridemia, hypercalcemia 8-Autoimmune Pancreatitis (AIP)

Clinical Features of chronic pancreatitis include

***Epigastric pain**

The pain is usually epigastric and may radiate to the back increases in a supine position and decreases on leaning forward. Pain can be intermittent or continuous, anorexia**,** and Weight loss, Painless chronic pancreatitis occurs in a few patients who present with malabsorption.

*__Malabsorption__: fat and protein malabsorption occur after the loss of 90% of the pancreatic secretory capacity.

***Diabetes -**pancreatic endocrine insufficiency results in glucose intolerance as insulin production

drops below requirements.

***Vit B12 malabsorption**

The physical findings

*Thin patient with Epigastric tenderness, skin pigmentation over the abdomen and back is common and results from chronic use of a hot water bottle [erythema ab igene].

*The presence of jaundice it might indicate associated biliary disease due to scarring in the pancreatic head.

*Palpable abdominal mass if Pancreatic pseudocysts exist.

Investigations

Blood test

-Serum amylase may be normal or maybe elevated in acute –on –chronic episodes.

-Low albumin, calcium, and B12 suggest Malabsorption

-Elevated ALP reflect biliary obstruction

-Impaired glucose tolerance test or diabetes.

Pancreatic function test

Stool fat-in advance pancreatic insufficiency stool fat may reach 30-40gr /day

Bentiromide test.

Imaging

A plain abdominal X-ray may show pancreatic calcification.

Us, CT, EUS which shows pancreatic abnormal lesion, calcifications, ERCP and MRCP revealing duct distortion, and side branch dilatation.

Complications

Pancreatic cancer, duodenal obstruction, portal, and splenic vein thrombosis, bile duct obstruction

Treatment.

1-Pain control

*Patients with severe and persistent pain should avoid dietary fat and alcohol

*****Narcotics and NSAID**.**

*Endoscopic treatment of chronic pancreatitis may involve sphincterotomy of the minor or major pancreatic sphincter, dilatation of strictures, removal of calculi, or stenting of the pancreatic duct.

*****Antioxidants, coeliac plexus block, surgical drainage, and resection

2- Malabsorption

*Pancreatic enzyme replacement therapy. with a proton pump inhibitor may be required (proton pump inhibitors prevent the acid breakdown of the pancreatic enzyme supplement).

*Oral fat restriction [with supplementary medium-chain triglyceride] which do not require lipase for digestion

3-Diabetes needs carbohydrate restriction and insulin therapy. coexisting deficiency of glucagon increased the risk of hypoglycemia.

Nutritional assessment is essential, as advanced Chronic pancreatitis is often associated with malnutrition.

Autoimmune Pancreatitis (AIP)

This is a form of chronic pancreatitis that can mimic cancer but which responds to corticosteroids. * Characterized by abdominal pain, weight loss, or obstructive jaundice.

*Blood tests reveal increased serum immunoglobulin G (IgG) or IgG4, and the presence of other autoantibodies.

*Imaging shows a diffusely enlarged pancreas, narrowing of the pancreatic duct and stricturing of the lower bile duct on ERCP. histologically by lymphoplasmacytic infiltrate.

AIP may occur alone or in association with other autoimmune disorders such as Sjögren's syndrome, primary sclerosing cholangitis (PSC), or inflammatory bowel disease. The response to steroids is usually excellent but some patients require azathioprine.

Pancreatic tumors

include

A-Exocrine pancreatic cancer

1-Pancreatic adenocarcinoma Arise from ductal and acinar cells. They are more common in men. Around 95% of pancreatic tumors are adenocarcinomas, nearly all of which are ductal adenocarcinomas. Risk factors for pancreatic carcinoma include-- chronic pancreatitis, smoking, old age, diabetes, obesity, diets high in meat and cholesterol, and inherited cancer syndromes including Peutz–Jegher syndrome. About 70% of pancreatic adenocarcinoma is located in the head and the remainder is in the body and tail. pancreatic cancer spreads locally by direct extension and to distance sites by lymphatic and vascular channels**.**

Clinical features

Tumors of the head produce symptoms earlier in contrast to tumors of body and tail

* Abdominal pain, often radiating to the back. Abdominal pain due to invasion of the celiac and superior mesenteric arterial plexus.

*Weight loss and anorexia.

*Obstructive jaundice caused by lesions in the head of the pancreas.

*Cancer in the body and tail may cause splenic vein obstruction causing splenomegaly, gastric and esophageal varices, and GI haemorrhage.

*Glandular destruction by the tumor may lead to diabetes and/or pancreatic exocrine insufficiency

Investigations

* Elevated ALP and bilirubin in bile duct obstruction. *CA 19-9 is not useful in diagnosis but, if positive, may be used to monitor response to treatment. *Helical CT or MRCP in the case of obstructive jaundice, *ERCP.

*Fine-needle aspiration using CT or endoscopic ultrasound guidance.

*PET is done with non-contrast CT to enhance anatomic details. The normal pancreas usually not seen by PET scan.

Management

*Surgery- pancreatotico-duodenectomy [Whipples operation].

*Adjuvant therapy - chemotherapy and radiotherapy after surgery to prevent cancer recurrence and improve survival.

*Neo-adjuvant therapy –given before surgery to a downstage tumor to facilitate surgery and to prevent distance metastasis.

*Pain control by oral opioid analgesics at sufficient dose or percutaneous EUS guided or operative splanchnic (coeliac) block may be used.

*Pruritus is treated by relieving obstruction with palliative surgery or endoscopic biliary stenting. Where this is not possible, cholestyramine or phenobarbital may be used.

*Pancreatic insufficiency is treated with pancreatic supplements.

2-Ampullary carcinoma.

Clinical presentation

*Anorexia, Weight loss,malasia, abdominal pain, progressive jaundice, iron deficiency anemia

Recurrent acute pancreatitis of no identifiable etiology may be the presentation in some patients.

Diagnosis

*UGI endoscopy and biopsy,

*EUS, MRI, MRCP, and CT.

Treatment

Early tumor confined to the ampulla not invading the muscularis propria can be resected endoscopically

Pancreatico-duodenectomy is the most effective and definitive treatment.

B-Cystic tumors of the pancreas,

1-Mucinous Cystadenomas.

Are cystic lesion commonly seen in the body and tail of the pancreas more common in the middle-aged women. May be asymptomatic or may be presented with abdominal pain Or maybe found incidentally by the abdominal US, CT examination.

Diagnosis

CT or EUS guided fine needle cyst fluid analysis shows [thick and mucoid material with low amylase and high CEA].

Treatment

Surgery –distal pancreatectomy

2-Serous cystadenoma

More common in the body and tail. More common in middle-aged women. Usually asymptomatic and discovered incidentally.

Diagnosis

CT image may show tiny cyst separated by septa giving them honeycomb appearance

The cystic fluid analysis revealed low viscosity, low CEA and negative cytology

Management

For symptomatic lesion -surgery.

3-Intra-ductal papillary-mucinous neoplasm [IPMN]

This rare tumor most commonly occurs in women, in the tail of the pancreas. The tumor over-secretes

mucin, causing pain and recurrent bouts of pancreatitis. The disease may range from benign to malignant and cannot be differentiated without surgical removal. diagnosis is made by CT, MRCP, or ERCP.
Following surgical resection, the prognosis is excellent for benign disease. The malignant disease has a 5-year survival of 50−75%.
Management- pancreatico-duodenectomy.

C-Pancreatic endocrine tumors [PET]

These rare tumors arise from the islet and gastrin-producing cells of the pancreas. They may be non-
functioning and present with biliary obstruction, bleeding or abdominal masses; or functioning (hormone
secreting) and present with an endocrine syndrome, depending on the hormone produced.

1-insulinoma

Rare tumors were more common in females. May part of MEN1

Presentation

*Intermittent confusion,sweating,nausea,vomiting,weakness,seizur, syncope or coma.

Investigation- shows Hypoglycemia<50mg% with high insulin level.

Diagnosis-

*During fasting glucose, insulin,
C -peptide level measured at 3-6 hrs interval.
*EUS

Treatment

*Dietary modifications.
*Diazoxide a non- diuretic thiazide has hyperglycemic effects.
*Octreotide.
*Surgery.

2-Gastrinoma is the second most commonly functional PET but gastrinomas are also frequently found outside the pancreas Up to 30% of gastrinomas are associated with multiple endocrine neoplasia type 1 (MEN-1), discussed at page 18-19.

3-Glucagonomas

Secrets an excessive amount of glucagon

Presentation

*Weight loss [due to catabolic effects of glucagon], Anemia [decrease erythropoiesis], necrolytic migratory erythema
*Venous thromboembolism

Investigations

*High glucose level
*High glucagon level, localization of tumor by CT

Management

Controlling hyperglycemia by insulin*Surgery*Octreotide for skin rash

4-Somatostatinoma

Secrete a large amount of somatostatin and cause a syndrome characterized by DM [secondary to the inhibitory action of somatostatin on insulin secretion], gallbladder disease, diarrhea, Steatorrhea, weight loss and achlorhydria

Diagnosis

EUS/FNA
Plasma somatostatin level

Treatment

Oral hypoglycemic drugs
Surgery.

5-VIPomas, [pancreatic cholera or WDHA]

WDHA-[Watery diarrhea, hypokalemia, achlorhydria**]**
A neuroendocrine tumor that secretes excessive amounts of Vasoactive intestinal polypeptide

VIP interact with intestinal epithelial cells leading to watery diarrhea. Achlorhydria results from inhibitory effects of the VIP hormone on HCL secretion. Hyperglycemia results in glycogenolytic effects of VIP on the liver. VIP-oma occurring in pancreas, retro peritoneum, liver and esophagus.

Clinical features

large volume secretory diarrhea that persists with fasting and flushing

differential diagnosis includes

gastrinoma, celiac disease, chronic laxative abuse

diagnosis

high fasting plasma VIP level, localizing of the tumor by CT, MRI, EUS

Treatment

*Fluid and electrolyte replacement

*Octreotide