Large bowel tumors

Learning objectives

Understanding the types of polyps and its role in colorectal cancer-1

Pathology of large bowel tumours-2

Clinical presentations-3

Investigations and management-4

Large bowel tumours:

Polyps and polyposis syndromes:

1- Polyps may be neoplastic or non-neoplastic “hamartomas, metaplastic (hyperplastic) polys and inflammatory”.

2- Polyps may be single or multiples. (Few mm-several cm).

3-Colorectal adenomas are extremely common in the western world and prevalence increasing with age and are more

. common in the rectum and distal colon

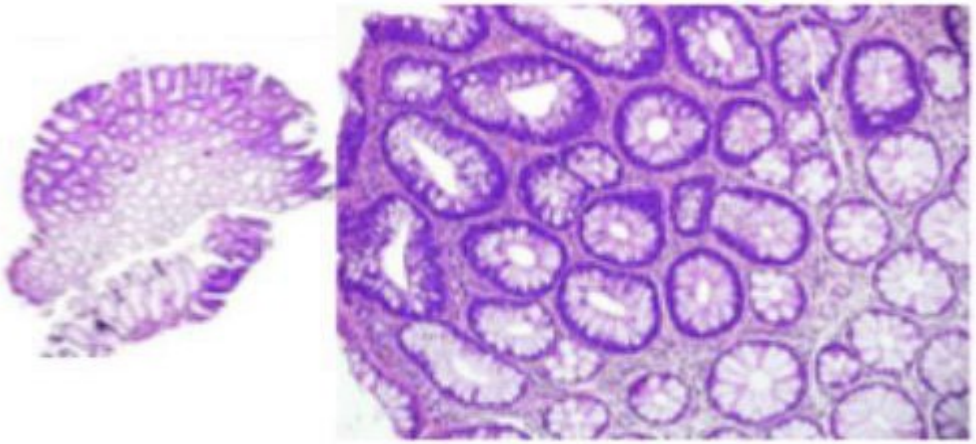
- Histologically, they are classified as either tubular, villous or tubulovillous according to glandular architecture.

- Nearly all forms of colorectal carcinoma develop from adenomatous polyps over 5-10 years.

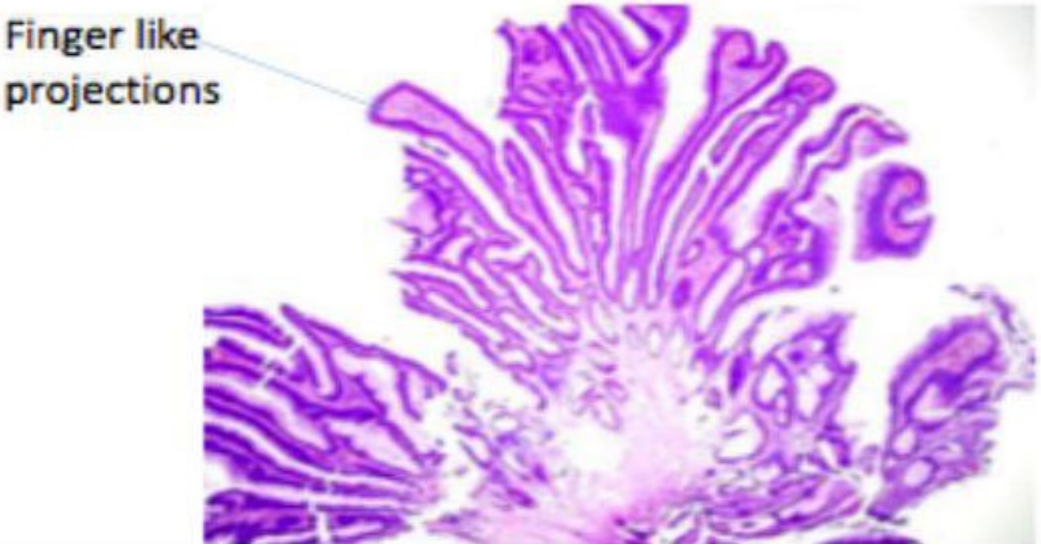
- Adenomas are usually asymptomatic.

- Discovery of a polyp at sigmoidoscopy is an indication for colonoscopy because proximal polyps present in 40-50%.

Tubular adenoma



Villous adenoma



Colonoscopic polypectomy reduces subsequent colorectal-

.cancer risk

-Surveillance colonoscopy should done at 3-5 years internal after all polyps removed.

- Patient >75 years of age do not require repeated colonoscopy.

Indication for segmental colonic resection in polyps:

1- Cancer cells found within 2mm of the resection margin of the polyp.

2- When the polyp cancer is poorly differentiated.

3-When lymphatic invasion is present

Familial adenomatous polyposis (FAP):

1- It’s an autosomal dominant.

2- (1/13.000) uncommon.

3- 1% of all colorectal cancer.

4- Result from germ line mutation of the APC gene on the long arm of chr. 5 or acquired mutation.

5- 20% of cases arise as new mutation (No family history).

6- 80% of these multiple polyps (hundreds to thousands) develop by age 15. With symptoms develop few years later.

.7- Cancer develop 10-15 years later

Extraintestinal features of FAP:

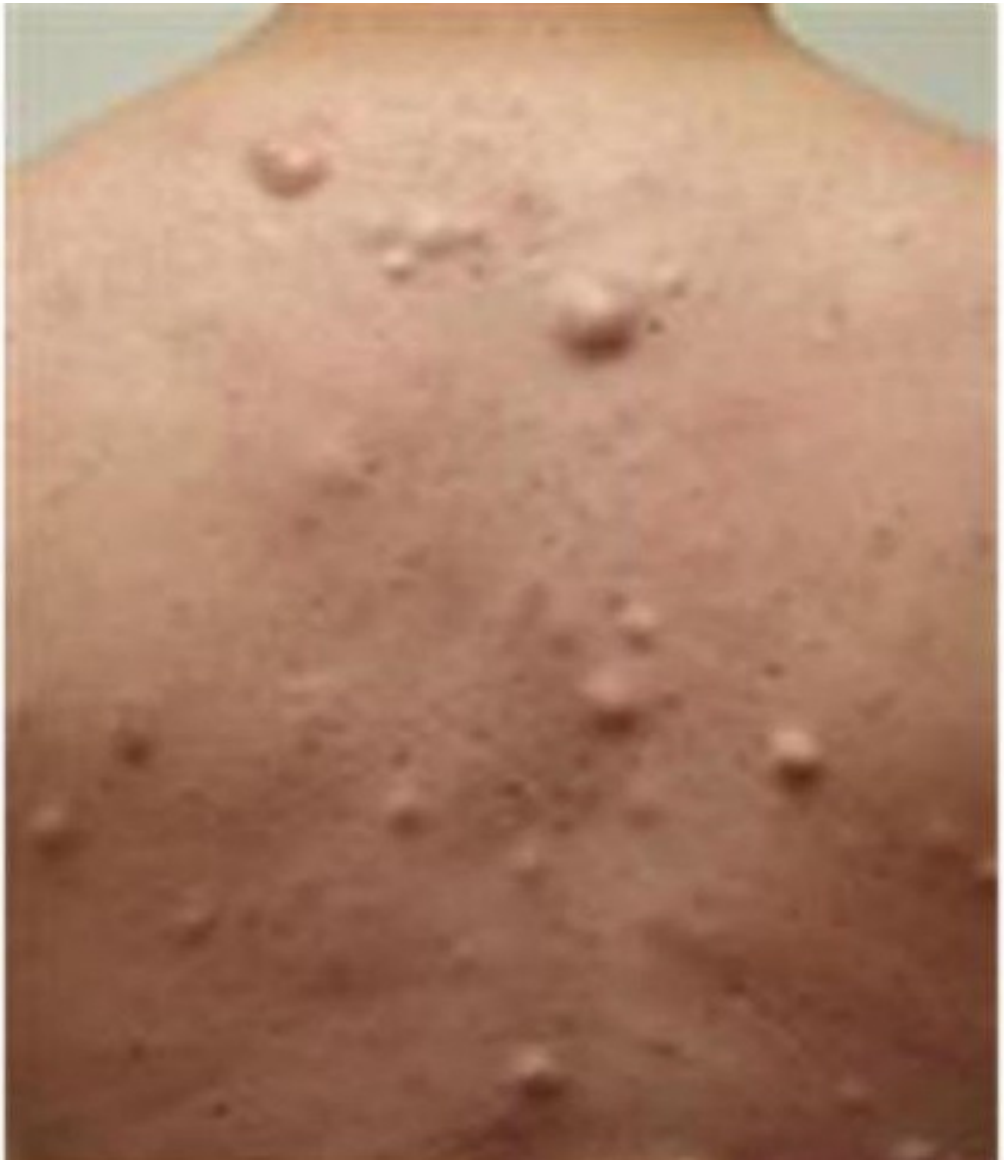
1- Subcutaneous epidermoid cysts (extrimities, face, scalp).

2- Benign osteomas (skull and angle of mandible).

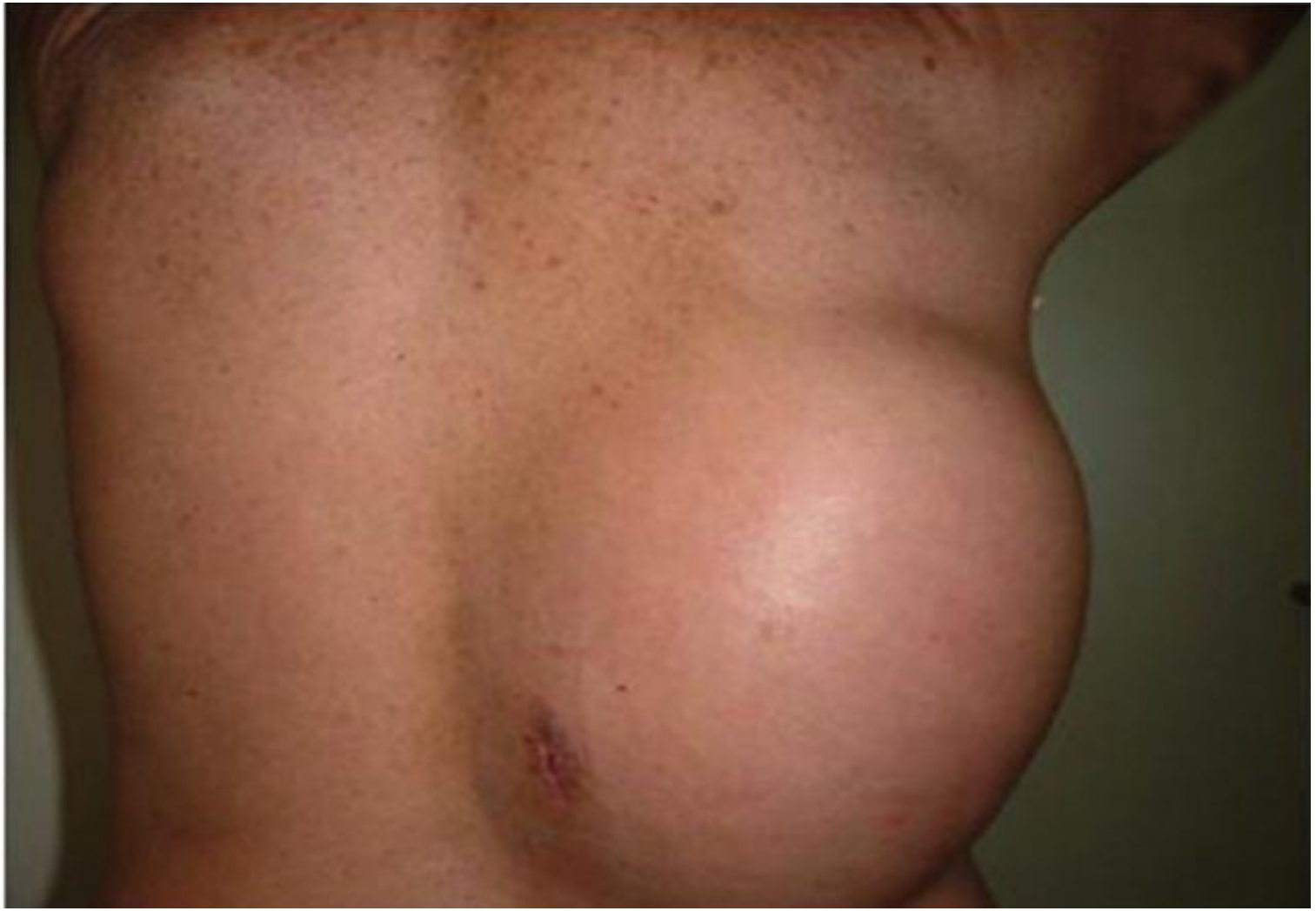
3- Congenital hypertrophy of the retinal pigment epith.

4- Desmoids tumours.

.5- Lipomas



Epidermoid cyst



Desmoid tumer

Diagnosis and management:

1- Sigmoidoscopy.

2- Genetic testing by DNA linkage analysis:

a- Done for newly diagnosed cases with new mutation and

.for all first degree relatives

b- And for families with FAP at 13-14 years of age. (regular sigmoidoscopy reserved for those known to have mutation). Affected individuals should undergo colectomy after school or college education has been completed (preserve anal part) followed by periods of upper scope to detect deudenal adenomas.

Peutz-Jeghers syndrome:

1- Hamartomatous polyp (multiple, affect small and large intestine).

2- Melanin pigmentation of lips, mouth and digits.

3- Asymptomatic or present with chronic bleeding, anaemia or intussusception.

4-Small but significant risk of small bowel adenocarcinoma and of cancer of pancrease, lung, ovary, breast, endometrium .

5- Mutation in serine – threonine kinase gene.

6- 30% inherited in an autosomal dominant manner.

.7- Up to 20% develop colorectal cancer > 40 years


Pigmented areas around the lips


Hamartomatous Polyp

Criteria for diagnosis:

1- ≥10 colonic Juvenile polyp.

2- Juvenile polyps elsewhere in the gut. OR

3- Any no. of polyps + family history.

Colorectal cancer:

1- Uncommon in the developing world.

2- In western countries: 2nd most common internal malignancy and 2nd leading cause of cancer deaths.

.3-Increase over 50 years of age

Pathology:

1- Most tumours arise from malignant transformation of a benign adenomatous polyp.

2- 65% ⎓ rectosigmoid.

3- Either polypoid and fungating or annular and constricting.

4- Spread occur through bowel wall.

5- Rectal cancers may invade the pelvic viscera and walls.

6- Lymphatic invasion is common at presentation “both portal ”.(hepatic) and systemic (lungs) circulation

C/F

1- Symptoms depend on the site.

Left colon ⎓ fresh blood + early obstruction.

Right colon ⎓ Anaemia (occult blood) + altered bowel habit + obstruction (late).

2- Colicy lower abdominal pain 2/3.

3- Rectal bleeding 50%.

4- Minority (obstruction or perforation ⇨ peritonitis, abscess, fistula).

5- Weight loss.

6- O.E/ may be palpable mass, anaemia or hepatomegaly. low rectal tumours may be palpable on PR.

Investigation:

1- Rigid sigmoidoscopy detect < 1/3 of tumours.

2- Colonoscopy is the investigation of choice.

3- Barium enema (less sensitive and specific than colonoscopy).

4- Endoanal Ult. or pelvic MRI. Or CT colography.

. 5-CEA (carcinoembryonic Ag)

Management:

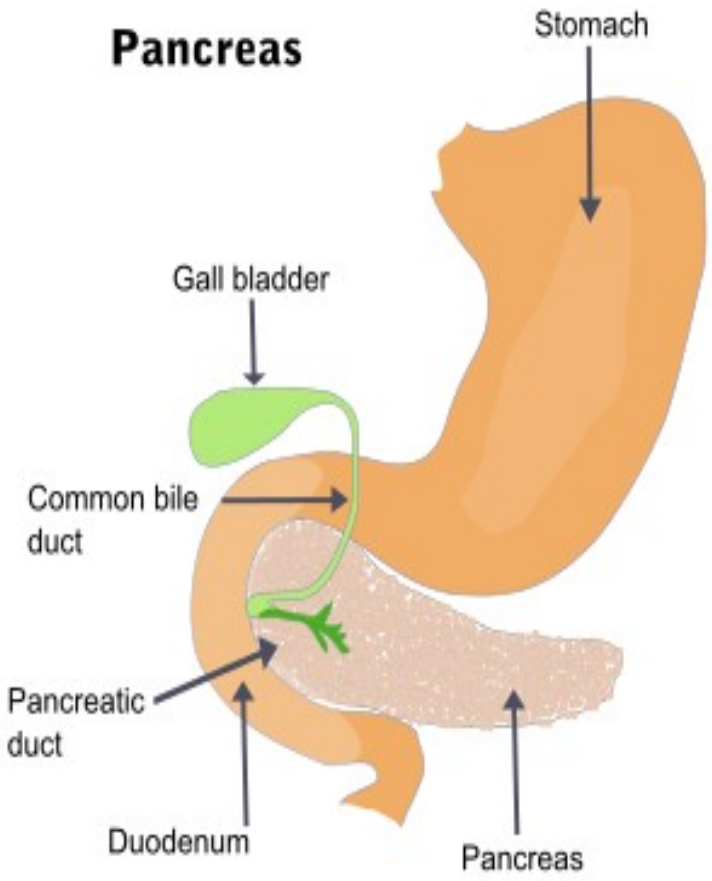
1- Surgery.

2- Adjuvant therapy.

a- 5- Fluorouracil + folic acid (for 5 months).

. b- Pre-operative radiotherapy

:Diseases of the Pancreas



Acute pancreatitis

- Learning objectives
- Pathophysiology-1
- Clinical presentations-2
- Complications-3
- Management-4
- Bad prognostic factors-5

Acute pancreatitis "pathophysiology"

1- 3% of all abdominal pain admitted to hospital.

2- Premature activation of zymogen granules, releasing proteases which digest the pancreas and surrounding tissue. .(CBD, dendenum, splenic vein and transverse colon)

3- Severity of acute pancreatitis depend upon the balance between proteolytic enzymes and antiproteolytic factors (intracellular pancreatic trypsin inhibitor protein and circulating B2 – macroglobulin, $\acute{\alpha}_1$- antitrypsin and C1- esterase inhibitors).

4- It is either mild and self limiting or sever with local complications (necrosis, pseudocyst or abscess and systemic complications leading to multi-organ failure). Mortality 30%.

C/F:

1- Sever- constant upper abdominal pain, radiate to the back, build up over 15-60 minutes.

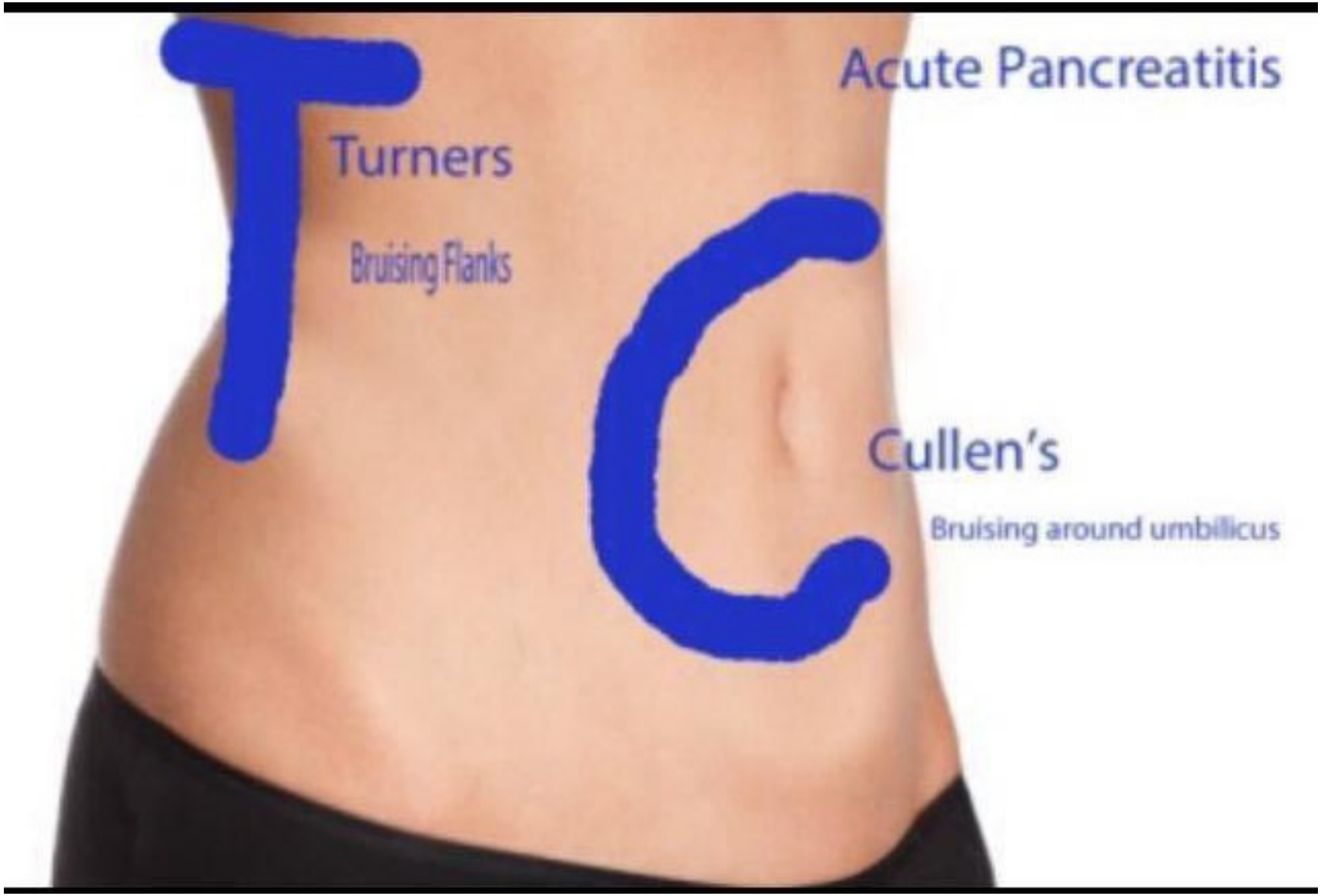
2- Nausea and vomiting are common.

3- Epig. Tenderness but absent guarding and rebound tenderness. “retroperitoneal”

4- Paralytic ileus.

5- Hypoxia and hypovolmic shock with oliguria.

6- Flanks discoloration (Grey Turner’s sign) or periumbilical region (Cullen’s sign) are features of sever pancreatitis and .haemrrhage


Acute Pancreatitis
T
Turners
Bruising Flanks
C
Cullen's
Bruising around umbilicus

D,Dx:

1- Perforated viscus.

2- Acute cholecytitis.

.3- Myocardial infarction

Causes:

1- Common: gallstone, alcohol, idiopathic, post – ERCP.

2 - Rare: post-surgery, trauma, drugs (azathioprin, thiazide, sod. Valproate), metabolic (hyper Ca^{+}, triglyceride), pancreas divisum, sphincter of oddi dysfunction, infection (mump, coxsackie virus), heridatory, renal failure (R.F), organ transplant, .sever hypothermia

Complications:

1- Systemic – systemic inflammatory response syndrome.

(Increase vascular permeability, paralytic ileus, vomiting, renal failure).

- Hypoxia (ARDS).

- Hyperglycemia.

- Hypocalcemia.

.- Decrease serum albumin

2- Pancreatic:

- necrosis.

- Abscess.

- Pseudocyst.

- Pancreatic ascetic or pleural effusion.

3- G.I:

- Upper GIT bleeding.

- Variceal haemorrhage.

- Colonic erosion.

- Deud. Obst.

- Obst. Jaundice.

Diagnosis:

1- Serum amylase or lipase.

2- Ultrasound or CT scan.

.3- Plain x-ray to exclude other diagnosis

Other causes of elevated serum amylase:

1- Intestinal ischemia.

2- Perforted peplic ulcer.

3- Ruptured ovarian cyst.

”4-Parotitis. “salivary

Management:

1- Establish the diagnosis.

2- Early treatment (resuscitation):

Analgesia using pethidine + correction of hypovolaemia (N.S or colloids).

Hypoxia ▭ O_2 or mechanical ventilation. In ARDS.

Hyperglycemia ▭ Insulin

Hypocalcaemia + tetany ▭ calcium injection.

N.G aspiration ▭ if paralytic ileus is present

.Enteral feeding

Low does heparin ▭ prophylactic against thromboembolism.

prophylactic broad spectrum antibiotic. (imipenem or cefuroxime).

Urgent ERCP (cholangitis or Jaundice + sever acute pancreatitis) .or MRCP in less sever cases

Detection and treatment of complication:

Nectrotising pancreatitis or pancreatic abscess requires urgent surgical debridment.

Pancreatic pseudocyst treated by drainage into the stomach, .deudenum or Jejunum. (Roux en Y)

Adverse prognotic factors in acute pancreatitis:

1-Age > 55 year.

2- Po2 < 8 kpa (60 mm Hg).

3- WBC > 15 x 10 9/L.

4- Alb. < 32g/L.

5- S. Ca^{+} < 2mmol/L (8mg/ dL).

6- Glucose > (180 mg/dL).

7- Urea > 45 mg/dL.

8- Alanine aminotransferase > 200 u/L.

. 9- LDH > 600 u/L

Chronic pancreatitis

- Learning objectives
- Causes-1
- Clinical presentations-2
- Investigations-3
- Management-4

Chronic pancreatitis:

Causes:

1- Calcific (Alcoholic or Tropical).

2- Obstructive (stenosis of ampulla of vater).

3- Pancrease divisum.

4- Cystic fibrosis.

5- Hereditary.

. 6- Idiopathic

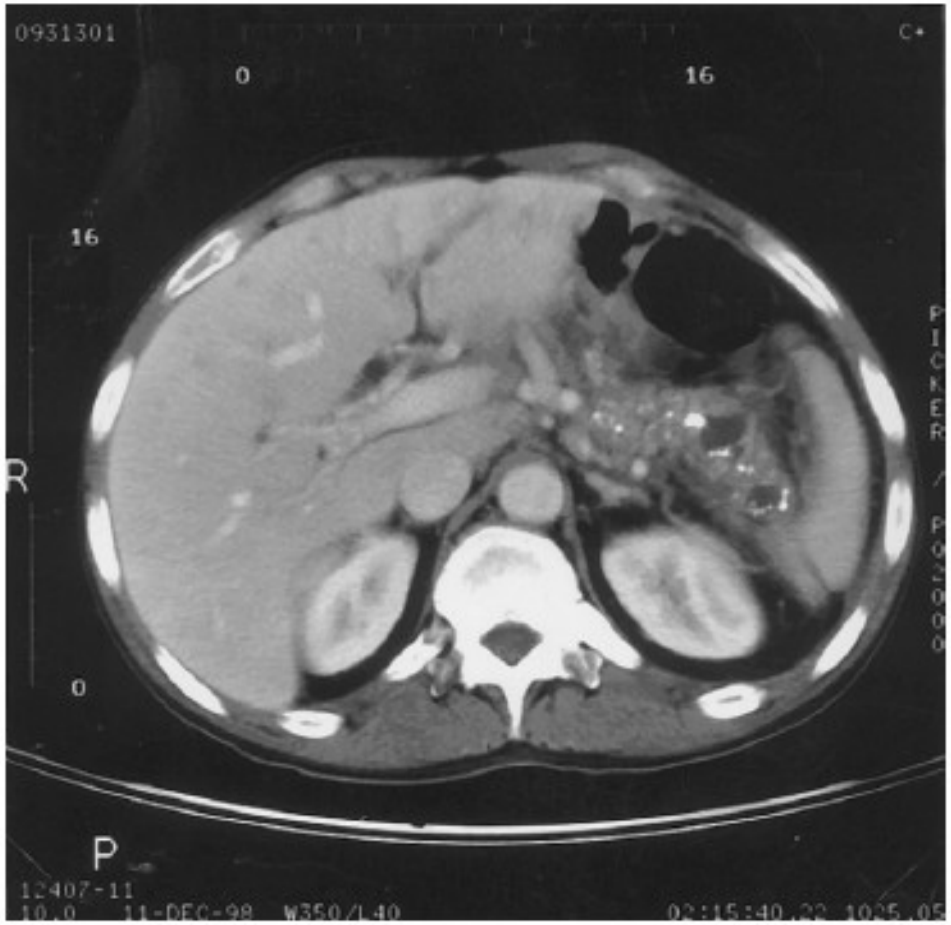


Figure 4 Computed tomography scan of a patient with calcifications and small pseudocysts in the pancreas consistent with chronic pancreatitis.

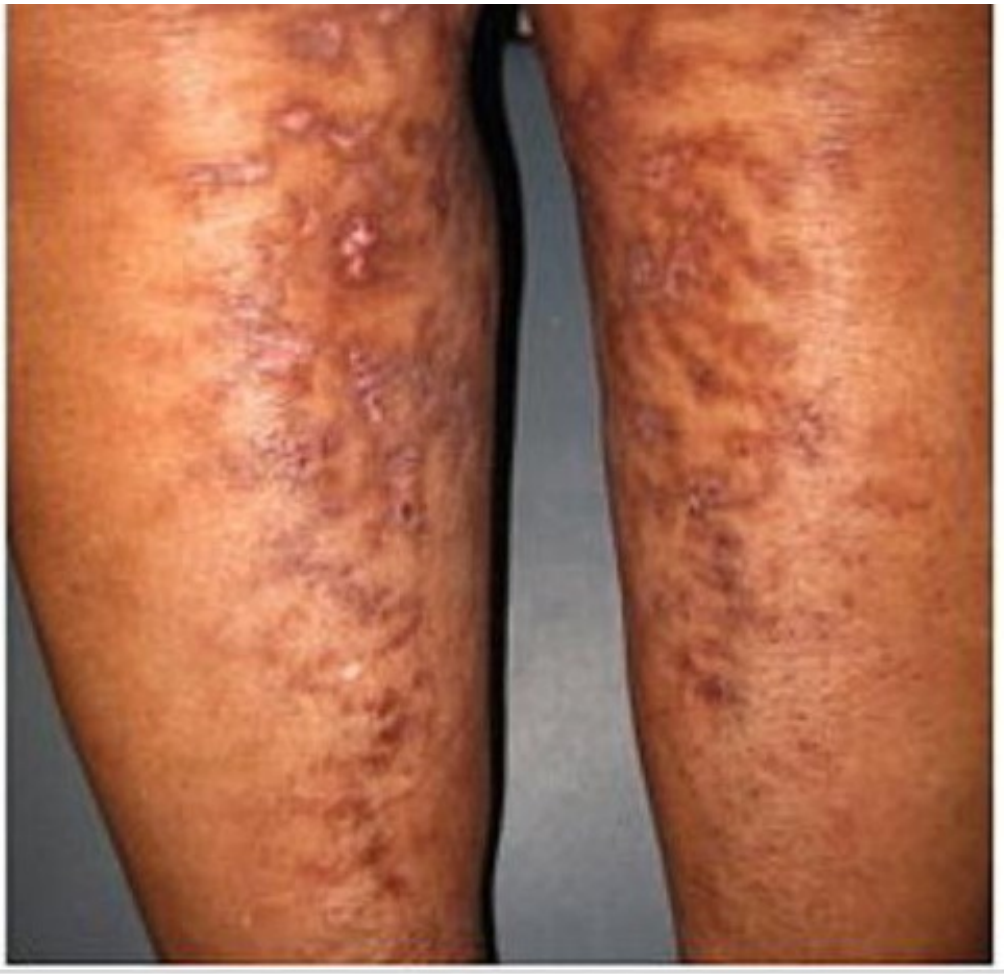
C/F:

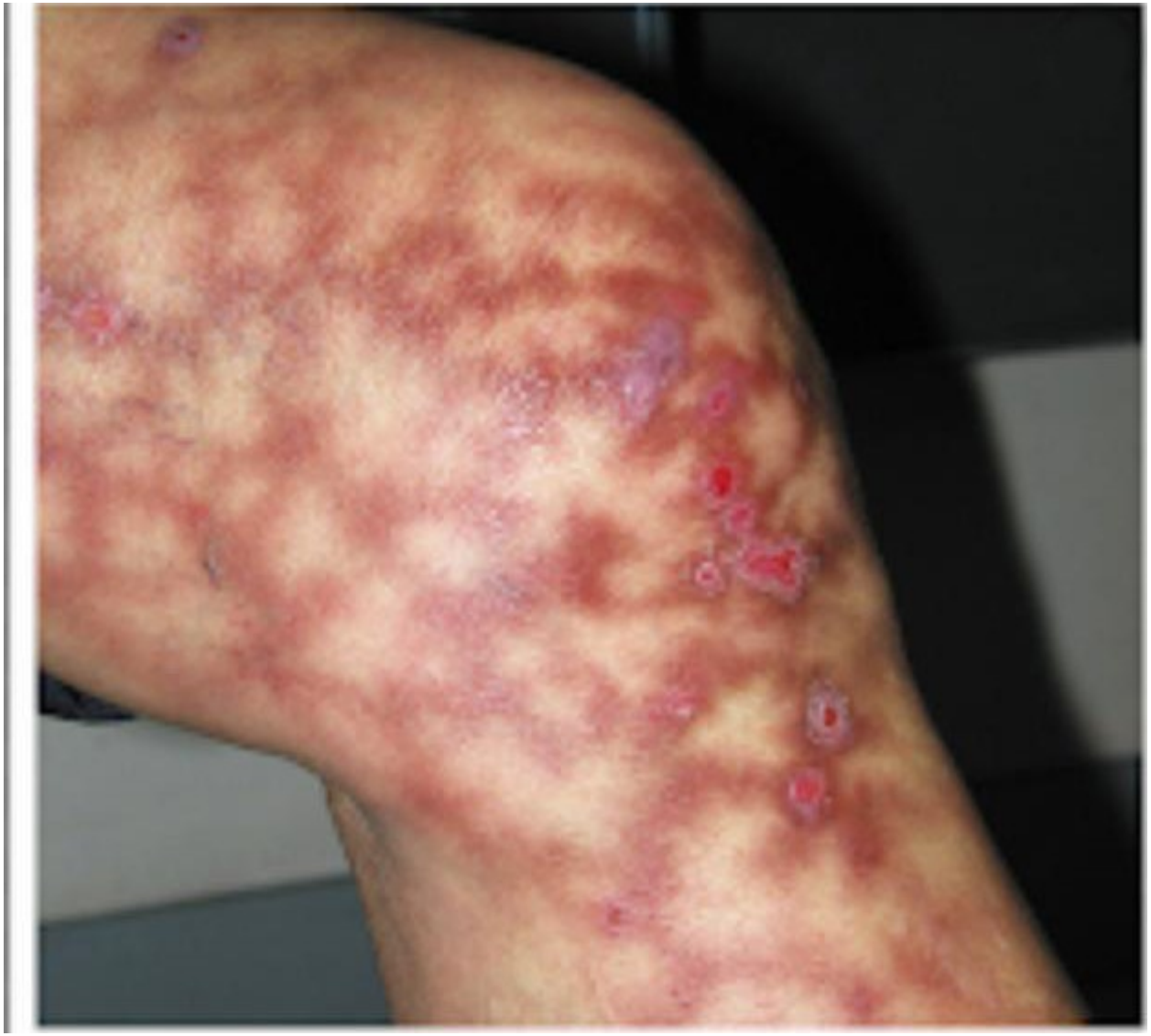
1- Abdominal pain (episodic or progressive or no pain but present with diarrhaea).

2 - Weight loss.

3- Steatorrhoea.

4- Erythema ab igne





Investigations:

1- Diagnostic tests: ultrasound, CT, x-ray, MRCP, endoscopic ultrasound.

2- Pancreatic function tests:

- pancreatic Juice collection following secretin injection.
- Pancreolauryl test.
- Fecal pancreatic chymotrypsin or elastase.
- Oral glucose tolerance test.

.3- Test of anatomy prior to surgery: MRCP

Management:

1-Alcohol avoidance.

2- Pain relief: NSAID, opiate, oral pancreatic enzyme supplements, celiac plexus neurolysis, total pancreatectomy.

3- Treatment of steatorrhoea: dietary fat restriction + oral pancreatic enzyme supplements ± proton pump inhibitor.

4- Treatment of complications: treatment of pseudocysts, .pancreatic ascitis, common bile duct or duodenal stricture