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Chapter 3

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 3

Gastroenterology

Energy from food • The amount of energy that may be derived from 1 gram of food is as follows: carbohydrates: 4 kcal protein: 4 kcal fat: 9 kcal • The amount of energy a food product contains is expressed in calories (kcal). In simple terms, per unit weight, fats contain twice as many calories as protein or carbohydrates.

Protein losing enteropathy Definition • excessive leakage of plasma proteins into the lumen of the GIT • refers to any condition of the GIT that results in a net loss of protein from the body. Causes • lymphatic obstruction, (lymphatic leakage secondary to obstruction.): e.g:
 primary intestinal lymphangiectasia,
 conditions associated with venous stasis such as right-sided heart failure. • mucosal disease: inflammatory exudation through mucosal damage: inflammatory bowel diseases,
 NSAID enteropathy,
 GI malignancy. increased permeability from non-erosive mucosal disease amyloidosis,
 GI infections, rheumatic diseases, Features • The most common presenting symptom is swelling of the legs due to decreased plasma oncotic pressure. bilateral oedema from hypoproteinaemia is generalised and may be seen in the periorbital region as well as in the extremities • diarrhoea Investigations • Measurement of α1-Antitrypsin in a sample of faeces
 the most appropriate to confirm the diagnosis Albumin is degraded by proteases in the gut; however, α1-antitrypsin is a plasma protein that is resistant to degradation by proteases (it is a protease inhibitor) and its measurement can indicate leakage of plasma proteins into the gut.
• Serum albumin albumin level <20 g/L (<2 g/dL) is usually required to cause peripheral oedema
 Finding of low serum albumin prompts investigation to determine whether the aetiology is due to loss in the urine, hepatic synthetic dysfunction, or gastrointestinal losses. History of significant diarrhoea in conjunction with ruling out alternative causes makes the diagnosis. Treatment • Treat the underlying disease.

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Enteral feeding

Definition • Enteral feeding = any route of feeding that utilizes the patient's GI tract to deliver appropriate nutrition (differs from parenteral nutrition, which delivers nutrition intravenously, completely bypassing the GI tract) Enteral nutrition VS parenteral nutrition
• Enteral nutrition is preferred to parenteral nutrition whenever feasible - if the GI tract is functional, use it - benefits include improved absorption, immunological benefits, and helps maintain a healthy and functional GI tract Routes of enteral feeding: • Short-term: nasogastric tubes Consider gastric feeding unless upper GI dysfunction (then for duodenal or jejunal tube) • Long-term (> 2–3 weeks): gastrostomy or jejunostomy tubes
 Gastric feeding > 4 weeks consider long-term gastrostomy gastrostomy tube: mainly used in cases of proximal gastrointestinal tract obstruction to facilitate feeding.
 If it withdrew accidentally, reinsertion of the tube as soon as possible would be the preferred action. However, it needs a good level of expertise to do this.
 Therefore, in this case, insertion of a Foley's catheter is the best practice as it is easy to do, and this should preserve the opening of the skin and anterior abdominal wall muscles until a someone experience enough is available to re-insert the gastrostomy tube.

How to check NG placement? • Check NG placement using aspiration and pH (check post pyloric tubes with AXR) The first line investigation to confirm correct placement of a nasogastric tube is
 pH testing of gastric aspirate using indicator paper
 If the pH is between 1 and 5.5 then this is confirmatory evidence of correct placement.
 If the pH reading is between 5.5 and 6 it is recommended that a second independent reading is made to confirm. if aspirate pH ≥6 nasogastric feeding tubes feeding cannot be commenced
 If there is any doubt, then an appropriately interpreted chest x ray is a second line investigation.
Key points • Identify patients as malnourished or at risk (see below) • Identify unsafe or inadequate oral intake with functional GI tract • Consider bolus or continuous feeding into the stomach • PEG can be used 4 hours after insertion but should not be removed until >2 weeks after insertion.
Indications • pre-operative
 Surgical patients due to have major abdominal surgery: if malnourished, unsafe swallow/inadequate oral intake and functional GI tract then consider pre-operative enteral feeding.
• ITU patients
 ITU patients should have continuous feeding for 16-24h (24h if on insulin) Consider motility agent in ITU or acute patients for delayed gastric emptying. If this doesn't work, then try post pyloric feeding or parenteral feeding.

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Contraindications • Mechanical ileus, bowel obstruction • Acute abdomen (e.g., severe pancreatitis, peritonitis) • Upper GI bleeding • Mucositis • Severe substrate malabsorption • Congenital GI anomalies • High-output fistulas • Nonfunctional GI tract (e.g., gastroschisis, short bowel syndromes)

Patients identified as being malnourished • BMI < 18.5 kg/m2 • unintentional weight loss of > 10% over 3-6/12 • BMI < 20 kg/m2 and unintentional weight loss of > 5% over 3-6/12 AT RISK of

malnutrition • Eaten nothing or little > 5 days, who are likely to eat little for a further 5 days • Poor absorptive capacity • High nutrient losses • High metabolism Causes of diarrhoea in patients receiving enteral nutrition: • hyperosmolar feed • bacterial contamination • low feed temperature • reduced intestinal absorptive capacity • too rapid or irregular administration • lactose intolerance. Causes of constipation in patients receiving enteral nutrition: • Inadequate fluid replacement

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Refeeding syndrome Refeeding syndrome hypophosphataemia

Definition:
• Refeeding syndrome describes the metabolic abnormalities which occur on feeding a person following a period of starvation (≥ 5 days).

Pathophysiology: • When malnourished, the body uses endogenous fuel stores for energy and maintains serum electrolytes by redistribution from intracellular spaces. • Exogenously administered glucose results in insulin release. This results in rapid uptake of glucose, potassium, phosphate and magnesium into cells, with dramatic falls in the extracellular concentrations.

Features • Hypophosphataemia (symptoms are due predominantly to hypophosphataemia,) • hypokalaemia • hypomagnesaemia • abnormal fluid balance • Due to understood reasons, the body retain fluid ↑ extracellular space ↑cardiac work acute heart failure.

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• neurological problems resulting in: Oedema Lethargy Confusion Coma Convulsions, and Death.

• Nausea and diarrhoea is also common due to gut intolerance. Prevention (NICE 2006) • Identify patients at a high-risk of developing refeeding syndrome Patients are considered high-risk: if one or more of the following:

1. BMI < 16 kg/m2
2. unintentional weight loss >15% over 3-6 months
3. little nutritional intake > 10 days
4. hypokalaemia, hypophosphataemia or hypomagnesaemia prior to feeding (unless high) If two or more of the following:
5. BMI < 18.5 kg/m2
6. unintentional weight loss > 10% over 3-6 months
7. little nutritional intake > 5 days
8. history of: alcohol abuse, drug therapy including insulin, chemotherapy, diuretics and antacids • Decrease oral calorific intake to less than 50% of the recommended amount. NICE recommend that if a patient hasn't eaten for > 5 days, aim to re-feed at no more than 50% of requirements for the first 2 days. limit initial dietary intake to 1000–1500 kcal/day
   Management • Correcting electrolyte abnormalities aggressively it may be preferable

to provide electrolyte replenishment prior to commencing calorific intake

A patient with a history of alcoholism is admitted for re-feeding. Which component of the feed may need to be reduced to avoid encephalopathy? Protein protein content of feeds should be strictly managed in patients with alcoholism. Protein rich feeds ↑ total ammonia burden ↑ risk of encephalopathy.

Melanosis coli

• Melanosis coli is a disorder of pigmentation of the bowel wall.
• Causes It is associated with laxative abuse, especially anthraquinone compounds such as senna This phenomenon is seen in over 70% of persons who use anthraquinone laxatives (for example, cascara sagrada, senna, and frangula) within several months of use. Also alternative "medicine" drugs contain ingredients like cascara which contain anthraquinones. The modern laxatives such as liquid paraffin and polyethylene glycol do not cause these changes. • Pathophysiology

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 Chronic use of anthraquinone laxatives cause injury to the colonic epithelium, with generation of lipofuscin pigment. This pigment is subsequently engulfed by the macrophages to give rise to the histological picture.
• Diagnosis Melanosis coli is a histological diagnosis made from rectal biopsy material which shows numerous macrophages filled with brown pigment within the lamina propria.
 Histology demonstrates pigment-laden macrophages The macroscopic appearance varies from deep black pigmentation to reticulated brown discolouration. • Treatment The condition is benign and reversible on stopping the laxatives.

Mesenteric ischaemia (ischaemic colitis)

The two most common symptoms of ischemic colitis are severe abdominal pain and hematochezia (passage of fresh blood through the anus).

• Mesenteric ischaemia is primarily caused by arterial embolism resulting in infarction of the colon.
• More likely occur in areas such as the splenic flexure that are located at the borders of the territory supplied by the superior and inferior mesenteric arteries. especially the superior mesenteric artery.

Predisposing factors • increasing age • atrial fibrillation • other causes of emboli: endocarditis • cardiovascular disease risk factors: smoking, hypertension, diabetes Features • abdominal pain abdominal pain exacerbated by eating is suggestive of mesenteric ischaemia. Pain that is disproportionately severe compared to the abdominal findings is characteristic.

• rectal bleeding • diarrhoea • fever • bloods typically show an elevated WBC associated with acidosis • Acute mesenteric ischaemia is a cause of elevated amylase that is unrelated to pancreatitis. • Elevated serum lactate also suggests ischaemic aetiology. Diagnosis • CT scanning: the imaging modality of choice, with a sensitivity and specificity over 90%. If the presentation is clearly of acute bowel ischaemia then a CT angiography would be the best test.
 the presentation is consistent with several other possible causes of bloody diarrhoea and abdominal pain (i.e. acute colitis), Flexible sigmoidoscopy would be the best investigation - safer than colonoscopy (relative contraindication in active colitis), allowing biopsies to be taken and the viewing of a possible pseudomembrane.
 Occasionally the mucosa has a characteristic appearance. Biopsies show ulceration and a polymorphonuclear infiltrate. Haemosiderin-laden macrophages are characteristic but uncommon. • Angiography: if the diagnosis is in doubt.

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• Mucosal edema can be seen as the thumbprinting sign on plain abdominal radiograph and barium enema. • Flexible sigmoidoscopy
 The finding of ulceration which spares the rectum is typical
 ulceration extending to the splenic flexure corresponds with the arterial supply of the inferior mesenteric artery.

Management • supportive care • balloon angioplasty and stenting the preferred treatment for hemodynamically stable patients with acute mesenteric ischemia who do not present with signs or symptoms of advanced intestinal ischemia (peritonitis, sepsis) because this procedure is minimally invasive and studies suggest similar efficacy to open surgical treatment.
• laparotomy and bowel resection laparotomy is reserved for acutely ill patients who are hemodynamically unstable or have evidence of peritonitis (rebound tenderness and involuntary guarding).

MRCPUK-part-1-jan-2018: Which part of the bowel is most prone to ischaemic colitis? Splenic flexure because it receives its blood supply from terminal branches of the superior mesenteric and inferior mesenteric arteries, creating a watershed area.

Small bowel bacterial overgrowth syndrome (SBBOS) Definition • (SBBOS) is a disorder characterised by excessive amounts of bacteria in the small bowel resulting in gastrointestinal symptoms of bloating, abdominal distension and diarrhoea .Risk factors for SBBOS • neonates with congenital gastrointestinal abnormalities • scleroderma • absent gastric acid secretion • small bowel diverticulae • fistulae between the small and large bowel • small bowel strictures • diabetes mellitus (diabetic neuropathy) • adhesions. Features: It should be noted that many of the features overlap with irritable bowel syndrome: • chronic diarrhoea • bloating, flatulence • abdominal pain • Biochemically there is classically a low vitamin B12 level and normal or elevated folate level as a result of bacterial metabolism of B12 to folate.

Steatorrhoea and flatulence are classic presenting features of small bowel bacterial overgrowth.

Investigation • The gold standard investigation of bacterial overgrowth is small bowel aspiration and culture • Other possible investigations include: hydrogen breath test

 14C-xylose breath test 14C-glycocholate breath test: used increasingly less due to low specificity • In practice many clinicians give an empirical course of antibiotics as a trial

Management • correction of underlying disorder • antibiotic therapy: rifaximin is now the treatment of choice due to relatively low resistance.
• Co-amoxiclav or metronidazole are also effective in the majority of patients.

Spontaneous bacterial peritonitis (SBP)

• (SBP) is a form of peritonitis usually seen in patients with ascites secondary to liver cirrhosis. most commonly seen in alcoholic cirrhosis • typically caused by aerobic gram negative bacteria. (usually Escherichia coli, Klebsiella) sponteanous bacterial peritonitis is almost without exception caused by a single organism. • Diagnosis paracentesis: neutrophil count > 250 cells/ul Sending some ascitic fluid in blood culture bottles increases the yield. high serum ascites albumin gradient (SAAG) (>11 g/L) ascitic fluid and the white cells will be predominantly neutrophils (>500 WBCs/mm3and >50% neutrophils). • Management: intravenous cefotaxime is usually given other option: IV piperacillin-tazobactam
 It is important to start antibiotics promptly pending the results of an ascitic analysis. Antibiotic prophylaxis should be given if: patients who have had an episode of SBP patients with fluid protein <15 g/l and either Child-Pugh score of at least 9 or hepatorenal syndrome Norfloxacin is recommended for short term prophylaxis. • Prognosis Alcoholic liver disease is a marker of poor prognosis in SBP. Has poor prognostic significance with a one-year survival after a diagnosis of between 30-50%. An episode of spontaneous bacterial peritonitis carries a two-year mortality rate of 50%. • Differential diagnosis pancreatic ascites (eg. Acute pancreatitis) elevated fluid amylase helps confirm this (particularly the characteristic way in which it is in excess of the serum value). The low lactate dehydrogenase (<225 IU/L) helps exclude a polymicrobial ascitic fluid infection which has similar findings
 no mention of finding any organisms on the Gram stain. Bacterial growth occurs in about 80% of specimens with polymorphonuclear (PMN) count of >250 cells/mm3. Ascitic fluid analysis demonstrates a low serum albumin ascites gradient (SAAG) (<11 g/L).

 Cirrhosis and spontaneous bacterial peritonitis are both characterised by a high SAAG (>11 g/L) and are differentiated from each other on the basis of white cell count, Gram stain and culture results. secondary bacterial peritonitis (ruptured viscus or loculated abscess).
 Lactate dehydrogenase >225mU/L, glucose <50mg/dL, total protein >1g/dL and multiple

organisms on gram stain suggest secondary bacterial peritonitis (ruptured viscus or loculated abscess).
 Chylous ascites
 A high level of triglycerides confirms chylous ascites.
 elevated amylase level suggest pancreatitis or gut perforation.
 elevated bilirubin level suggest biliary or gut perforation.

Abdominal tuberculosis (Tubercular peritonitis) Features • risk of tuberculosis
• should always be suspected in the severely malnourished patient • Constitutional symptoms are common, including fever, anorexia and weight loss. • extensive lymphadenopathy.
Investigations • The cut-off for considering ascitic fluid to be exudative would be 30 g/l, but in the setting of hypoproteinaemia, this is less relevant.
• The marked increase in white cell count is strongly supportive of a diagnosis of infective ascites.
Diagnosis • The most sensitive test to establish the diagnosis is visually directed (laparoscopic) peritoneal biopsy with histology and culture for TB. • Although PCR of ascitic cells/fluid has increased non-invasive diagnosis, the best yield remains from laparoscopy and peritoneal biopsy, which in recent series led to a diagnosis in 95% of cases. • An alternative in this setting might be to perform fine needle aspiration or excision biopsy of one of the palpable lymph nodes. • The diagnostic yield of ascitic culture for mycobacteria is very low (<10%) even with closed culture systems.

Which investigation is most likely to yield a diagnosis? Laparoscopy and peritoneal biopsy

VIPoma

VIP (vasoactive intestinal peptide) • source: small intestine, pancreas • stimulation: neural • actions: stimulates water and electrolytes secretion by pancreas and intestines,
 inhibits gastric acid and pepsinogen secretion peripheral vasodilation ,
 potentiates acetylcholine action on salivary glands. VIPoma: WDHA syndrome Watery Diarrhea, Hypokalemia, Achlorhydria.

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VIPoma • 90% arise from pancreas • large volume diarrhoea, secretory diarrhoea ('pancreatic cholera') The normal daily stool weight is 250–300 g
 A stool volume of <700 mL/d excludes the diagnosis of VIPoma. What is the most likely mechanism of diarrhoea? Secretory due to enterocyte stimulation • weight loss • dehydration • hypokalaemia, hypochlorhydria. Achlorhydria • hypokalaemic acidosis (loss of alkaline secretions) • mildly raised glucose. • raised plasma pancreatic polypeptide • abdominal colic • cutaneous flushing • raised plasma VIP

Volvulus • Volvulus defined as torsion of the colon around it's mesenteric axis resulting in compromised blood flow and closed loop obstruction. • Sigmoid volvulus (around 80% of cases) describes large bowel obstruction caused by the sigmoid colon twisting on the sigmoid mesocolon. A similar problem may also occur at the caecum (20% of cases).
• In most people (around 80%) the caecum is a retroperitoneal structure so not at risk of twisting. In the remaining minority there is however developmental failure of peritoneal fixation of the proximal bowel putting these patients at risk of caecal volvulus.

Sigmoid volvulus associations Caecal volvulus associations • older patients • chronic constipation • Chagas disease • neurological conditions e.g. Parkinson's disease, Duchenne muscular dystrophy • psychiatric conditions e.g. schizophrenia all ages adhesions pregnancy

Features • constipation • abdominal bloating • abdominal pain • nausea/vomiting Diagnosis • usually diagnosed on the abdominal film The most helpful early diagnostic tool of intestinal obstruction is the plain abdominal X-ray. • sigmoid volvulus:
 large bowel obstruction (large, dilated loop of colon, often with air-fluid levels) + coffee bean sign (omega sign) • caecal volvulus:
 small bowel obstruction may be seen

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Sigmoid volvulus
The most important feature of a sigmoid volvulus rather than a large redundant distended loop of sigmoid colon is the absence of haustra.

Management • sigmoid volvulus:
 rigid sigmoidoscopy with rectal tube insertion • caecal volvulus:
 management is usually operative. Right hemicolectomy is often needed

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Imaging in bowel obstruction Looking for small and large bowel obstruction is one of the key indications for performing an abdominal film.

Small bowel Large bowel Maximum normal diameter = 35 mm

Valvulae conniventes extend all the way across Maximum normal diameter = 55 mm

Haustra extend about a third of the way across

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Small bowel obstruction

CT from a patient with small bowel obstruction secondary to adhesions. Distension of small bowel loops proximally (duodenum and jejunum) with abrupt transition to intestinal segment of normal caliber. Presence of small amount of free fluid intracavity.

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Radiology: pneumoperitoneum • An erect chest x-ray is a useful investigation in patients with an acute abdomen as it may demonstrate free air in the abdomen (pneumoperitoneum) - an abnormal finding suggestive of a perforated abdominal viscus (e.g. a perforated duodenal ulcer). • Rigler's sign (double wall sign) may be seen on an abdominal film. CT is now the preferred method for detecting free air in the abdomen.

Erect chest x-ray with air visible under the diaphragm on both sides.

Abdominal x-ray demonstrates numerous loops of small bowel outlined by gas both within the lumen and free within the peritoneal cavity. Ascites is also seen, with mottled gas densities over bilateral paracolic gutters. In a normal x-ray only the luminal surface (blue arrows) should be visible outlined by gas. The serosal surface (orange) should not be visible as it is normally in contact with other intra-abdominal content of similar density (other loops of bowel, omentum, fluid). In this case gas abuts the serosal surface rendering it visible. As this film has been obtained i (t b f i fl id l l) it l i th li tt ith fl id i d i

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Dumping syndrome
• occur in up to 50% of patients who have undergone gastric bypass when high levels of simple carbohydrates are ingested. • early dumping syndrome rapid onset , usually within 15 minutes of eating
 results from rapid emptying of food into the small bowel. Due to the hyperosmolality of the food there are rapid fluid shifts from the plasma into the bowel leading to hypotension and a sympathetic nervous system response.
 The presenting symptoms are often colicky abdominal pain, diarrhoea, nausea, and tachycardia.
 Treatment: usually self-limiting and resolves within 7 to 12 weeks. Patients should avoid foods high in simple sugar and replace them with high fibre, complex carbohydrates and protein-rich foods. Small, frequent meals
 leaving a 30 minute gap between solids and liquids
• Late dumping syndrome
 occurs as a result of the hyperglycaemia and subsequent insulin response leading to hypoglycaemia which takes place two to three hours after a meal. Symptoms include dizziness, fatigue, sweating, and weakness.
 Management is similar to early dumping syndrome.

Small bowel lymphoma
Pain is the most common presenting feature of small bowel lymphoma • Lymphoma comprises 15-20% of all small bowel tumours with the ileum most commonly affected. • Primary lymphomas of the small bowel include mucosa-associated lymphoid tissue (MALT) lymphoma diffuse large B cell lymphoma immunoproliferative small intestinal disease (IPSID), and enteropathy-associated T cell lymphoma (EATL). • Patients with coeliac disease are at higher risk of T cell lymphoma. • There is a male predominance • the median age at presentation of 25 years.
• Patients may present with: anorexia weight loss nausea and vomiting chronic pain abdominal fullness early satiety, and constipation. Findings on CT vary and may include multiple tumours, narrowing of the bowel lumen and mesenteric nodal masses.

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Pancreatic conditions Acute pancreatitis

• acute inflammation of the pancreas release of exocrine enzymes auto-digestion.
Pathophysiology • Sequence of events leading to pancreatitis: Intrapancreatic activation of pancreatic enzymes: secondary to pancreatic ductal outflow obstruction (e.g., gallstones, cystic fibrosis) or direct injury to pancreatic acinar cells (e.g., alcohol, drugs) Enzymatic autodigestion of pancreatic parenchyma Attraction of inflammatory cells (neutrophils, macrophages) → release of inflammatory cytokines → pancreatic inflammation (pancreatitis) • Sequelae of pancreatitis (depending on the severity of pancreatitis) Capillary leakage: Release of inflammatory cytokines and vascular injury by pancreatic enzymes → vasodilation and increased vascular permeability → shift of fluid from the intravascular space into the interstitial space (third space loss) → hypotension, tachycardia→ distributive shock Pancreatic necrosis: Uncorrected hypotension and third space loss → decreased organ perfusion → multiorgan dysfunction (mainly renal) and pancreatic necrosis
 Hypocalcemia: Lipase breaks down peripancreatic and mesenteric fat → release of free fatty acids that bind calcium → hypocalcemia

Causes

Popular mnemonic is GET SMASHED • Gallstones account for 50% of cases, with the majority of the rest being associated with alcohol. For prediction of a biliary etiology, an ALT level has the highest positive predictive value of any biochemical test. • Ethanol Amylase/lipase levels are markedly elevated in gallstone pancreatitis (thousands), whereas less increased in alcoholic (hundreds)
 raised mean corpuscular volume (MCV) suggests chronic high alcohol use • Trauma • Steroids • Mumps (other viruses include Coxsackie B) • Autoimmune (e.g. polyarteritis nodosa), Ascaris infection • Scorpion venom • Hypertriglyceridaemia, Hyperchylomicronaemia, Hypercalcaemia,Hypothermia • ERCP (acute pancreatitis following ERCP should be treated with I.V fluids + analgesia) • Drugs (azathioprine, mesalazine, didanosine, bendroflumethiazide, furosemide, pentamidine, steroids, sodium valproate) pancreatitis is 7 times more common in patients taking mesalazine than sulfasalazine Hypertriglyceridaemia (with level > 10 mmol/l) is a

risk factor for acute pancreatitis
The aetiology of acute pancreatitis should be determined in at least 80% of cases and no more than 20% should be classified as idiopathic The commonest causes in UK are gallstones and alcohol

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Hypertriglyceridaemia • Definitions: hypertriglyceridaemia > 1.7 mmol/L.
 Severe hypertriglyceridaemia >11.2-22.4 mmoL/L
 very severe as > 22.4 mmol/L. • The third commonest cause of acute pancreatitis after alcohol and gallstones.
• Considered a risk factor for pancreatitis when triglyceride levels are above 11.2 mmol/L. • In a patient with hypertriglyceridaemia and acute abdominal pain, an amylase should be checked to exclude acute pancreatitis.
Features • Patients typically present with severe epigastric pain which radiates to the back, and vomiting.
• there is often a systemic inflammatory response (SIRS)
• Serum amylase is classically raised three or more times normal,
• hypocalcaemia is relatively common.
• Raised bilirubin and/or serum aminotransferase suggest underlying gallstones.
• Cirrhosis results in a small shrunken liver and raised ALT and ALP (and gamma-GT if the cause is alcohol).
• Rare features associated with pancreatitis include: ischaemic (Purtscher) retinopathy - may cause temporary or permanent blindness • Skin changes (rare)
 Cullen's sign: periumbilical ecchymosis and discoloration (bluish-red)
 Grey Turner's sign: flank ecchymosis with discoloration
 Fox's sign: ecchymosis over the inguinal ligament Marker of severity
• CRP is now a widely used marker of severity in acute pancreatitis.
• Other methods which have to correlate with prognosis include the Ranson criteria and APACHE II score Prognosis • Criteria of poor prognosis There are a number of scoring systems which can be used to guide prognosis, but they are unreliable within the first 48 hours of the illness. Ranson's scoring system reflect prognosis associated with acute pancreatitis. • Ranson's criteria on admission that signify a worse prognosis include: Criteria present at 0 hours: Age >55 years old - 1 point WBC >16 ×109 - 1 point Glucose >11.1 mmol/L - 1 point LDH >350 U/L - 1 point AST >250 U/L - 1 point Criteria present at 48 hours: Hematocrit fall of 10% or greater - 1 point Urea rise of 1.8 mmol/L or more despite fluids - 1 point Serum Calcium <2 mmol/L - 1 point pO2 <60 mmHg - 1 point (PaO2 of < 8.0 kPa) Base deficit >4 meq/L - 1 point Fluid sequestration >6000 mL - 1 point • The mortality associated with severe acute pancreatitis 20%
 often due to sepsis or multiorgan failure.
• Hematocrit (Hct)

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 Should be conducted at presentation as well as 12 and 24 hours after admissions ↑ Hct (due to hemoconcentration) indicates third space fluid loss and inadequate fluid resuscitation ↓ Hct indicates the rarer acute hemorrhagic pancreatitis • The following portend a poor prognosis in patients with acute pancreatitis: WCC

> 15 Urea 16 Calcium <2.0 Glucose 10 CRP 150

Complications • ARDS (adult respiratory distress syndrome),
• acute kidney injury
• disseminated intravascular coagulation (DIC). due to pancreatic enzymes entering the blood and acting on coagulation factors, thereby activating them. • Pancreatic pseudocyst Investigations • lab Tests to confirm clinical diagnosis Amylase is markedly raised, often in excess of four times the normal value. nonspecific Lipase: if ≥ 3 x the upper reference range → highly indicative of acute pancreatitis More specific and preferred for the diagnosis
 The enzyme levels are not directly proportional to severity or prognosis Tests to assess severity Hematocrit (Hct) Should be conducted at presentation as well as 12 and 24 hours after admissions ↑ Hct (due to hemoconcentration) indicates third space fluid loss and inadequate fluid resuscitation ↓ Hct indicates the rarer acute hemorrhagic pancreatitis WBC count Blood urea nitrogen ↑ CRP and procalcitonin levels
 ↑ ALT • Images Ultrasound the most useful initial test Main purpose: detection of gallstones and/or dilatation of the biliary tract (indicating biliary origin) Signs of pancreatitis Indistinct pancreatic margins (edematous swelling)

 Peripancreatic build-up of fluid ; evidence of ascites in some cases Evidence of necrosis, abscesses, pancreatic pseudocysts CT with contrast
 not routinely indicated only when the diagnosis is in doubt would be preferable to ultrasound in establishing the presence of inflammation (acute or chronic) of the pancreas and severity of disease
 Abdominal x ray
 has NO role in acute pancreatitis
 Sentinel loop sign: dilatation of a loop of small intestine in the upper abdomen (duodenum/jejunum)
 Colon cut off sign:
 gaseous distention of the ascending and transverse colon that abruptly terminates at the splenic flexure
 Evidence of possible complications:
 pleural effusions,
 pancreatic calcium stones;
 helps rule out intestinal perforation with free air may demonstrate calcification in chronic

pancreatitis.
Follow-up: • All patients with persistent symptoms and greater than 30% pancreatic necrosis, and those with smaller areas of necrosis and clinical suspicion of sepsis, should undergo image guided fine needle aspiration to obtain material for culture 7–14 days after the onset of pancreatitis
Treatment
• supportive, and monitoring (often in the intensive care unit).
 Fluid resuscitation: aggressive hydration with crystalloids (e.g., normal saline)
 Analgesia: IV opioids (e.g., fentanyl) Bowel rest (NPO)and IV fluids are recommended until the pain subsides
 Nasogastric tube insertion:
 not routinely recommended;
 indicated in patients with vomiting and/or significant abdominal distention Nutrition Begin enteral feeding (oral/nasogastric/naso-jejunal) as soon as the pain subsides Total parenteral nutrition:
 only in patients who cannot tolerate enteral feeds (e.g., those with persistent ileus and abdominal pain) • if there is gallstones: urgent ERCP when stable.
 All should have a cholecystectomy either during the same admission or within four weeks depending on their clinical progress.

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Systemic inflammatory response syndrome (SIRS) Causes • sepsis
• pancreatitis Criteria
• SIRS is defined as two or more of the following:

1. Temperature more than 38°C or less than 36°C
2. Heart rate more than 90 beats/min
3. Respiratory rate more than 20 breaths/min or PaCO2 less than 4.3 kPa
4. WBC count 12,000/mm3, less than 4000/mm3 , or more than 10% immature (bands) form. Management • resuscitation of the sick patient still follows the ABC algorithm:
5. Airway
6. Breathing
7. Circulation. Airway control and oxygen to maintain normal saturations is the first part of that algorithm.
   Subsequent fluid resuscitation and treatment of the underlying cause can then be initiated.
   The need for invasive monitoring and intensive care is then assessed, depending on the response to initial treatment. • Early goal-directed therapy (EGDT) in cases of SIRS or septic shock is becoming increasingly recognised as potentially beneficial.
   EGDT aims to: increase organ perfusion through restoration of mean arterial pressure using inotropes if necessary,
   maintaining central venous pressure (CVP),
   maintaining oxygenation using SjVO2 (jugular venous oxygen saturation) as a guide to oxygen utilisation at the tissue level.
   If fluids are not achieving haemodynamic stability, and there is hypoperfusion

(indicated by oliguria or lactataemia) the most appropriate course of action central line vigorous resuscitation is indicated. Insertion of a central line allows measurement of CVP, SjVO2 and the use of inotropes. SjVO2 higher than 70% is indicative of organ hypoperfusion, as oxygen is not being extracted. • Obtain blood cultures prior to antibiotic administration

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Pancreatic pseudocysts Definition • encapsulated collection of pancreatic fluid which develops 4 weeks after an acute attack of pancreatitis; can occur in both acute and chronic pancreatitis Pathophysiology • pancreatic secretions leak from damaged ducts → inflammatory reaction of surrounding tissue → encapsulation of secretions by fibrous tissue Clinical features • Often asymptomatic • Painless abdominal mass • Pressure effects • Gastric outlet obstruction (early satiety, non-bilious vomiting, abdominal pain)

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• Bile duct obstruction with jaundice Diagnostics
• abdominal ultrasound/CT/MRI → extrapancreatic fluid collection withinwell-defined wall/capsule, no solid cyst components detectable Treatment • Surgical/endoscopic; ultrasound/CT-guided percutaneous drainage

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Chronic pancreatitis

Definition • Chronic pancreatitis is an inflammatory condition, which can ultimately affect both the exocrine and endocrine functions of the pancreas. Causes • alcohol excess (80%)
 what is the general mechanism by which alcohol induces the likely condition? Alcohol increases acinar cell sensitivity to CCK (cholecystokinin), stimulating trypsinogen production in the cell • Unexplained (20%) • PRSS-1 mutation can cause a hereditary form of the disease.
 It does this by allowing trypsin to be activated in the pancreas, thus causing enzymatic damage.
• SPINK-1 mutation can cause a hereditary form of the disease.
 It does this by allowing trypsin to be activated in the pancreas, thus causing enzymatic damage.
Features • pain is typically worse 15 to 30 minutes following a meal • steatorrhoea:
 symptoms of pancreatic insufficiency usually develop between 5 and 25 years after the onset of pain Late manifestation (after 90% of the pancreatic parenchyma is destroyed) • diabetes mellitus develops in the majority of patients. It typically occurs more than 20 years after symptom begin Investigation • abdominal x-ray shows pancreatic calcification in 30% of cases. • CT is more sensitive at detecting pancreatic calcification.
 Sensitivity is 80%, specificity is 85% More sensitive in moderate to advanced chronic pancreatitis
 Malabsorption is only present in moderate to advanced chronic pancreatitis abnormalities include: pancreatic calcification,
 pseudocyst formation and
 ductal distortion.

 CT scanning is much less effective in the diagnosis of early chronic pancreatitis and a normal scan does not exclude the diagnosis. • functional tests: faecal elastase may be used to assess exocrine function if imaging inconclusive • Both 72-hour faecal fat estimation and D-xylose absorption testing are used for their ability to indicate the presence, or absence, of malabsorption, neither is diagnostic of an underlying condition. associated with normal urinary D-xylose test findings Management • pancreatic enzyme supplements
 Pancrelipase (Creon)
• Analgesia

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 In a patient with chronic liver disease presented with features of decompensation associated with chronic pancreatitis Naloxone
 Patients with alcoholic liver disease are often surprisingly sensitive to opiate analgesia which should only be used with caution. • antioxidants: limited evidence base - one study suggests benefit in early disease

Complications • Pancreatic pseudocysts • Splenic vein thrombosis Occur in 10% of patients with chronic pancreatitis Pathophysiology: inflammation of the splenic vein → thrombus formation → leftsided portal hypertension → gastric varices
 Clinical features: can present with upper GI bleeding, ascites, and splenomegaly Diagnosis: ultrasound with doppler, CT/MR angiography Treatment Acute: anticoagulation and/or thrombectomy Chronic and symptomatic: splenectomy

• Pancreatic ascites • Pancreatic diabetes
• Pancreatic cancer (especially in patients with hereditary pancreatitis)

Pancreatic cancer • Pancreatic cancer is often diagnosed late as it tends to present in a non-specific way. • Over 80% of pancreatic tumours are adenocarcinomas • typically occur at the head of the pancreas. most often found in the ductal cells in the head of the pancreas. Associations • increasing age • smoking • diabetes • chronic pancreatitis (alcohol does not appear an independent risk factor though) • hereditary non-polyposis colorectal carcinoma • multiple endocrine neoplasia • BRCA2 gene • Jewish or African descent. Features • classically painless jaundice • however, patients typically present in a non-specific way with anorexia, weight loss, epigastric pain • loss of exocrine function (e.g. steatorrhoea) • atypical back pain is often seen the first symptom is often pain that radiates to the back. because it is found very late when it has already impinged on other structures. • migratory thrombophlebitis (Trousseau sign) is more common than with other cancers Migratory thrombophlebitis causes recurrent tender, palpable small blood clots that come and go in various locations on the body,
Investigation • ultrasound has a sensitivity of around 60-90% • high resolution CT scanning is the investigation of choice if the diagnosis is suspected • Carbohydrate Antigen 19-9 (CA-19-9) is a tumour marker is usually used to monitor response to treatment and possible recurrance, rather than for diagnosis. Management • less than 20% are suitable for surgery at diagnosis

Gastroenterology • a Whipple's resection (pancreaticoduodenectomy) is performed for resectable lesions in the head of pancreas.
 Side-effects of a Whipple's include dumping syndrome and peptic ulcer disease • adjuvant chemotherapy is usually given following surgery • ERCP with stenting is often used for palliation relief of symptoms as soon as possible is the main objective of therapy. Stenting relieves symptoms of itching and reverses jaundice in about 85% of patients.
 Stents can be inserted during an ERCP or percutaneously in those with extensive disease or in those otherwise unsuitable for surgery. Prognosis • It has a very high mortality rate (approximately 1 year from diagnosis), usually because it is found very late when it has already impinged on other structures.

Biliary conditions Ascending cholangitis • Ascending cholangitis is a bacterial infection of the biliary tree.
• The most common predisposing factor is gallstones. Features • Charcot's triad (occurs in about 20-50% of patients)

1. right upper quadrant (RUQ) pain, (70%)
2. fever (the most common feature, seen in 90%)
3. jaundice (60%) • hypotension and confusion are also common Combining these two additional symptoms to Charcot’s triad results in Reynold’s pentad. • elevated alkaline phosphatase and elevated direct bilirubin suggest obstruction of the biliary tree Investigation • The initial imaging study is ultrasonography. • The gold standard for diagnosis is (ERCP) endoscopic retrograde cholangiopancreatography. Management • intravenous antibiotics • endoscopic retrograde cholangiopancreatography (ERCP) after 24-48 hours to relieve any obstruction

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Gallstones (Cholelithiasis) Risk factors for biliary stones • Cholesterol gallstones are thought to arise as a result of a triple defect:

1. Super saturation of gallbladder bile (high in cholesterol, low in bile salts)
2. Increased rate of cholesterol nucleation in the gallbladder
3. Reduction in gallbladder contractility • Predisposing factors to gallstone formation: Older age Female sex (oestrogens) Oral contraceptive use Cirrhosis (bile pigment stones) ileal resection (by reducing entero-hepatic circulation and increasing bile salt loss)

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 Clofibrate (by increasing biliary supersaturation) rapid weight loss Cholestyramine (by binding bile salts) Crohn's disease Features • most will be asymptomatic • Classic symptoms include biliary colic, nausea, and/or vomiting biliary colic: sharp, colicky pain made worse with fatty food due to ↑ release of CCK contraction of gallbladder Investigation • liver function tests :

obstructive jaundice • Ultrasound abdominal/right upper quadrant ultrasound is the test of choice for gallstone disease ultrasound finding of a common bile duct dilatation is suggestive of an obstructing stone Whilst ultrasound is a good preliminary investigation for common bile duct stones it lacks sensitivity. The sensitivity of ultrasound for detecting stones is significantly reduced during an episode of acute pancreatitis (around 70%) so repeating an ultrasound is a reasonable suggestion as it would perform better in the current clinical context than it had done previously. However, its ability to detect CBD stones remains poor. MRI is highly effective in confirming the presence of common bile duct stones, endoscopic ultrasound (EUS) is a suitable alternative. CT does not perform well when compared to MRI. • Radiographs cannot rule out stone with negative radiograph because cholesterol stones are radiolucent pigment stones are radiopaque so may show up on radiograph • Endoscopic retrograde cholangiopancreatography (ERCP), along with intra-operative cholangiography, is considered the gold standard for diagnosis of common bile duct stones. However it is an invasive procedure associated with significant morbidity; thus it should ideally be performed as a therapeutic rather than diagnostic procedure. The indication for ERCP is for the removal of ductal stones (predominantly CBD stones). • Magnetic resonance cholangiopancreatography (MRCP) The presence of a CBD calculus should be confirmed prior to subjecting the patient to a potentially dangerous procedure such as an ERCP - MRCP would be the most appropriate test to do this. the most sensitive for a diagnosis of gallstones In terms of sensitivity for determining the presence of stones anywhere within the biliary tract, MRCP and EUS would be the most sensitive investigations with little to choose between them (ERCP may well miss small stones in the gallbladder). Management • In patients with severe gallstone pancreatitis ERCP and endoscopic stone extraction should be performed within 72 hours of the onset of pain. • In patients with mild gallstone pancreatitis, in the absence of cholangitis, there is no evidence to support ERCP and stone extraction in the acute setting; however arrangements

must be made for definitive management of common bile duct stones on the same admission or within two weeks of recovery.
• Asymptomatic gallstones which are located in the gallbladder are common and do not require treatment.
• However, if stones are present in the common bile duct there is an increased risk of complications such as cholangitis or pancreatitis and surgical management should be considered. • endoscopic retrograde cholangiopancreatography (ERCP) for biliary sphincterotomy and stone extraction. the most common procedure-related complication is Pancreatitis
 risks of developing this complication: Female sex,
 age less than 60 and
 a low probability of structural disease (suggested by a normal bilirubin, non-dilated ducts or suspected sphincter of Oddi dysfunction) • Percutaneous transhepatic cholangiography is an interventional radiological procedure which is generally reserved for therapeutic decompression of an obstructed biliary system where ERCP is unsuccessful or not possible.

Complications • Cholecystitis • Acute pancreatitis • Gallbladder cancer • Choledocolithiasis calculi in the common bile duct • Fistula between gallbladder and small intestine passed gallstone can obstruct the ileocecal valve Glasgow score for Pancreatitis:

1. PaO2 <7.29 kPa
2. Glucose >10 mmol/L
3. Age >55 years
4. WBC >15
5. Calcium <2.0 mmol/L
6. Urea >16 mmol/L
7. LDH >600 IU/L
8. Albumin <32 g/L Interpretation of glasgow score for pancreatitis: • The presence of three or more of these criteria within the first 48 hours is indicative of severe pancreatitis. • If the score ≥3, severe pancreatitis is likely Referral to the HDU/ICU is suggested in this case. If the score <3, severe pancreatitis is unlikely.

Functional gall bladder pain
• The Rome III criteria for functional gall bladder pain are as follows: episodes lasting 30 minutes or longer recurrent symptoms occurring at different intervals (not daily) the pain builds up to a steady level the pain is moderate to severe enough to interrupt the patient's daily activities or lead to an Emergency Department visit the pain is not relieved by bowel movements

 the pain is not relieved by postural change the pain is not relieved by antacids, and exclusion of other structural disease that would explain the symptoms. • The pain may present with one or more of the following supportive criteria: associated with nausea and vomiting radiates to the back and/or right infra subscapular region, and awakens from sleep in the middle of the night.

Choledochal cysts • Choledochal cysts are congenital bile duct anomalies, cystic dilatations of the biliary tree • The classic triad in adults with choledochal cysts is:

1. abdominal pain, (Most common symptom)
2. jaundice, and
3. palpable right upper quadrant abdominal mass.
   However, this triad is found in only 10-20% of patients. • Adults may present with complications (eg, hepatic abscesses, cirrhosis, portal hypertension, recurrent pancreatitis, cholelithiasis) • Abdominal ultrasonography is the investigation of choice • Choledochal cysts are usually diagnosed in the neonatal period but a few are delayed until adulthood. The Todani classification is used to define these: Type 1 - a fusiform dilation of the common hepatic duct (CHD) - the most common Type 2 - a diverticulum of the CHD Type 3 - a choledochcele Type 4 - describes extension into the intrahepatic ducts (the second most common) Type 5 - intrahepatic cystic disease only. • Treatment

 Resection and reconstruction is advised to prevent recurrent cholangitis, pancreatitis, and malignant change.

Sphincter of Oddi dysfunction • Type 1 Sphincter of Oddi dysfunction (SOD) is characterised by: abdominal pain,
 deranged liver function tests,
 a dilated biliary tree without strictures, and
 delayed emptying of contrast at ERCP.
 Delayed excretion of contrast is definitive and Sphincter of Oddi manometry need not be carried out with this finding. • Type 2 SOD pain with only one or two other criteria from the type 1 definition
• type 3 SOD biliary type pain only.
 Diagnosis in type 3 is supported by abnormal manometry although this will only be present in 12-28% of these patients so the diagnosis is most often one of exclusion.

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Gastroenterology

Post-cholecystectomy syndrome • Post-cholecystectomy syndrome is a recognised complication of cholecystectomies.
• Typically, symptoms of dyspepsia, vomiting, pain, flatulence and diarrhoea occur in up to 40% patients post-surgery. • The pathology behind the syndrome isn't completely clear, however there is some association with remnant stones and biliary injury.
• Pain is often due to sphincter of Oddi dysfunction and the development of surgical adhesions.
• Management: low-fat diet
 bile acid sequestrants, such as Cholestyramine, to bind the excess bile acids and thus preventing lower gastrointestinal signs.
 Proton-pump inhibitors like Lansoprazole do play a role, if the patient is complaining of dyspeptic like symptoms.

Bile-acid malabsorption

• Although a small proportion of bile acids (3%) are excreted in the faeces, about 97% of bile acids are recycled.
• Bile-acid malabsorption is a cause of chronic diarrhoea.
 the bile, with no gall bladder to store it, is excreted directly into the gut diarrhoea
• In people with bile acid malabsorption, excess bile in the colon stimulates electrolyte and water secretion, which results in chronic watery diarrhoea.
• May affect 10% of patients following cholecystectomy. • Typically it is post-prandial • There is evidence suggesting that up to one-third of people with a diagnosis of IBS with diarrhoea (IBS-D) have bile acid malabsorption

mechanisms
• Bile acid malabsorption causes diarrhoea by 1 of the following mechanisms:
 inducing secretion of sodium and water increasing colonic motility
 stimulating defecation inducing mucus secretion
 damaging the mucosa, thereby increasing mucosal permeability.

Types: divided into 3 types depending on aetiology:
• type 1: following ileal resection, disease or bypass of the terminal ileum
• type 2: primary idiopathic malabsorption
• type 3: associated with cholecystectomy, peptic ulcer surgery, chronic pancreatitis, coeliac disease or diabetes mellitus.

Causes:

1. Primary: due to an excessive production of bile acid,
2. Secondary: Due to an underlying gastrointestinal disorder, causing reduced bile acid absorption often seen in patients with ileal disease, such as with Crohn's. cholecystectomy coeliac disease