GKT X OSOC IS HELD IN NHH LECTURE THEATRE 2 - THE REST ARE ALL ONLINE VIA THE LINK IN OUR BIO!

PRESENTING OUR

GKTEACH

MSA Education

STAGE 2 SERIES

RESPIRATORY CONDITIONS	TUESDAY 14TH NOV 6:30PM
CARDIAC CONDITIONS	THURSDAY 16TH NOV 6:30PM
HAEMATOLOGICAL CONDITIONS	TUESDAY 21ST NOV 6:30PM
MENTAL HEALTH CONDITIONS	THURSDAY 23RD NOV 6:30PM
GASTRO CONDITIONS	TUESDAY 28TH NOV 6:30PM
GKTEACH X OSOC: CMS	THURSDAY 30TH NOV 6PM
METABOLIC CONDITIONS	TUESDAY 5TH DEC 6:30PM
ANATOMY FOR PT'S	THURSDAY 7TH DEC 6:30PM
PHARMACOLOGY FOR PT'S	TUESDAY 12TH DEC 6:30PM
COMMON GP CONDITIONS	THURSDAY 14TH DEC 6:30PM
LUCK DIP SBA SESSION	TUESDAY 19TH DEC 6:30PM

Gastroenterology + Hepatology for PTs

Aribah Naveed – Year 5
Tuesday 28 November 2023

Learning Outcomes

By the end of this session, you should be able to:

- Identify and differentiate between common gastrointestinal and hepatic disorders
- Explain the pathophysiological mechanisms underlying common gastrointestinal and hepatic disorders
- Identify appropriate diagnostic tools and tests for the evaluation and diagnosis of common gastroenterological and hepatological conditions

MSA Education

Some feedback from last session

- More questions for practice
- Pace was a bit slow
- Not explaining what conditions you're going to be covering in the talk because it makes it easy to guess the exact condition in each question
- Shorter pauses when waiting for people to give answers
- Maybe adding something interactive like mentimeter?

MSA Education

Notes

- More SBAs compared to the last session
  - Setup is: questions first, explanations after
- Faster pace (may not go through all the details of a particular condition)
- There will be lots of information on slides that you can look through later (slides will uploaded to the website: www.gktmsa.org)
- Can unmute or post questions in the chat

- P.s. do not worry if you do not know the answer ☺️

SBA

A 79-year-old lady is admitted from her residential home with signs of severe dehydration secondary to a 2-day history of severe vomiting and diarrhoea. She suffers from advanced dementia and is unable to provide a clear history. Her carers report she has been passing watery diarrhoea 7 times a day with occasional vomiting. They have not noticed any blood. She has been unable to tolerate fluids or solids. Several fellow residents have also had similar symptoms.

What is the most likely organism responsible for her symptoms?

A) Shigella

B) Bacillus cereus

C) Escherichia coli strain O157

D) Norovirus

E) Yersinia enterocolitica

A) Shigella

•Associated with outbreaks such as children attending school

B) Bacillus cereus

•Rapid onset diarrhoea after a meal
•Found in reheated rice

C) Escherichia coli strain O157

Associated with outbreaks of bloody diarrhoea

D) Norovirus

•Typically causes outbreaks of non-bloody vomiting and diarrhoea in healthcare settings

E) Yersinia enterocolitica

•Typically causes traveller's diarrhoea and often presents with intense right iliac fossa pain

Gastroenteritis

Bacterial	Viral	Parasitic
Staphylococcus aureus (cooked meats and cream)	Rotavirus: most common cause of infantile gastroenteritis	Cryptosporidium
Bacillus cereus (reheated rice)	Norovirus: most common cause of viral infectious gastroenteritis in all ages in England and Wales	Entamoeba histolytica
Clostridium perfringens (reheated meat, cooked meat)	Adenoviruses: commonly cause infections of the respiratory system but can also cause gastroenteritis, particularly in children	Giardia intestinalis
Campylobacter (uncooked meat)		Schistosoma
E.coli		
Salmonella (food)		
Shigella (outbreaks)		

Gastroenteritis

Conservative management
Strict hygiene and handwashing

Antibiotics if:
- systemically unwell
- immunosuppressed
- elderly

Microorganism	Antibiotic
Salmonella, shigella	Ciprofloxacin
Campylobacter	Macrolide e.g. erythromycin
Cholera	Tetracycline e.g. doxycycline

SBA

A 23-year-old girl presents to the General Practitioner due to several weeks of abdominal pain and non-bloody diarrhoea. The pain is colicky and more severe around the right iliac fossa (RIF). She describes experiencing frequent cramping abdominal pains and diarrhoea after meals. On further questioning, she reveals that she has lost 7 kg over the past few months. She has no relevant past medical history.

On examination, the patient is very slim and looks pale. Inspection of her mouth reveals aphthous ulcers. The abdomen is tender, particularly in the RIF. A mass can be felt in the RIF. Heart rate 70 bpm, BP 100/75 mmHg, temperature 36.8. There are several tender, erythematous nodules on her shins.

Which of the following is the most likely diagnosis?

A) Crohn's disease
B) Irritable bowel syndrome
C) Caecal carcinoma
D) Ulcerative colitis
E) Appendicitis

SBA

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C) Caecal carcinoma
D) Ulcerative colitis
E) Appendicitis

SBA

A 32-year-old female patient presents to the GP with a 4-week history of bloody diarrhoea and abdominal pain. She has no recent travel history or infective contacts, and no past medical history of note.

On physical examination there is mild diffuse abdominal tenderness. There is no peri-anal disease. Full blood counts shows a haemoglobin of 10.4 g/dL (low). Faecal calprotectin is raised.

Which of the following investigations findings is consistent with the most likely diagnosis?

A) Colonoscopy reveals multiple mucosal outpouchings

B) Colonoscopy reveals discontinuous involvement (skip lesions) and cobble stoning mucosa

C) Magnetic resonance angiography reveals obstruction of mesenteric vasculature

D) Colonoscopy reveals continuous mucosal inflammation, diffuse erythema, and loss of vascular markings

E) Raised faecal leukocytes

MSA Education

A) Colonoscopy reveals multiple mucosal outpouchings

•These colonoscopy findings are consistent with diverticular disease. This more typically presents in elderly patients with constipation and lower left sided abdominal pain

B) Colonoscopy reveals discontinuous involvement (skip lesions) and cobble stoning mucosa

•These colonoscopy findings are consistent with Crohn's disease. Crohn's disease is more likely to present with non-bloody diarrhoea, weight loss, and peri-anal disease

C) Magnetic resonance angiography reveals obstruction of mesenteric vasculature

•These findings are typical of mesenteric ischaemia. This is more likely to present in elderly patients with risk factors for cardiovascular disease

D) Colonoscopy reveals continuous mucosal inflammation, diffuse erythema, and loss of vascular markings

•This is the correct presentation. The clinical features are consistent with ulcerative colitis. Colonoscopy and biopsy is necessary for the diagnosis of ulcerative colitis

E) Raised faecal leukocytes

•Raised faecal leucocytes is suggestive of a bacterial infective colitis. The negative travel history and lack of sick contacts makes an infective colitis unlikely

Inflammatory bowel disease (IBD)

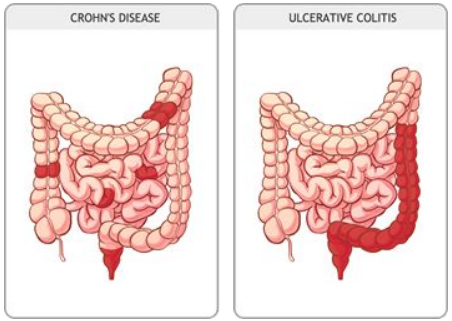
Crohn's disease

- Patchy transmural (affecting all layers of wall) granulomatous inflammation
- Distribution anywhere from mouth to anus
- Pain often in right iliac fossa
- Blood in stool less common
- Increased risk in smokers

Ulcerative colitis

- Continuous inflammation affecting mucosa only
- Distributed around rectum and colon
- Pain commonly in left iliac fossa
- Blood in stool more common
- Decreased risk in smokers

Crohn's disease	Ulcerative colitis
Crampy abdominal pain	Diarrhoea often containing blood +/- mucus
Diarrhoea	Tenesmus or urgency
Weight loss	Weight loss
Fever	Fever



Extra-intestinal manifestations

Crohn's disease	Ulcerative colitis
Dermatological manifestations: Erythema nodosum (painful erythematous nodules/plaques on the shins) Pyoderma gangrenosum (a well-defined ulcer with a purple overhanging edge)	Dermatological manifestations: erythema nodosum, pyoderma gangrenosum
Ocular manifestations: Anterior uveitis (painful red eye with blurred vision and photophobia) Episcleritis (painless red eye).	Ocular manifestations: anterior uveitis, episcleritis, conjunctivitis
Musculoskeletal manifestation: Arthritis (typically asymmetrical and non-deforming) Sacro-iliitis (similar to Ankylosis spondylitis).	Musculoskeletal manifestations: clubbing, non-deforming asymmetrical arthritis, sacroiliitis
Hepatobiliary manifestations: Gallstones (these are more common in Crohn's disease than in ulcerative colitis)	Hepatobiliary manifestations: primary sclerosing cholangitis

Endoscopic and histological descriptions

Crohn's disease	Ulcerative colitis
Macroscopic: aphthous ulcers, cobblestone appearance, skip lesions, rose thorn ulcers	Macroscopic: Continuous, uniformly inflamed mucosa erythematous, friable mucosa abnormal vascular pattern ulceration Inflammatory polyps ('pseudopolyps')
Microscopic: lymphoid hyperplasia, non-caseating granulomas, transmural inflammation	Microscopic: crypt abscesses and decreased globlet cell abundance.

SBA

A 24-year-old man has been experiencing right sided abdominal pain, diarrhoea and weight loss. On examination, his abdomen is tender in the right lower quadrant, and rectal examination reveals skin tags around the anus. His blood tests show: Hb 112g/L, WCC 11x10^9/L, platelets 350x10^9/L, CRP 100mg/L. A colonoscopy is normal.
What is the best test to confirm the most likely diagnosis?

A) Laparotomy
B) MRI small bowel
C) Gastroduodenoscopy
D) Abdominal x-ray
E) Faecal calprotectin

Crohn's management

To induce remission: steroids

Maintaining remission:

- azathioprine, mercaptopurine
- Methotrexate
- Biologics (adalimumab, infliximab)

Surgery is not curative

SBA

The 32-year-old female patient from the question before is admitted to the medical ward due to a flare-up of her ulcerative colitis. She reports that she opened her bowels seven times yesterday with notable blood in her stool. On examination, she has a temperature of 38.1°C with a pulse rate of 91 beats per minute. Routine blood tests show a raised erythrocyte sedimentation rate (ESR).

What is the most appropriate management plan?

A) Topical aminosalicylates

B) High-dose oral aminosalicylates

C) Intravenous corticosteroids

D) Emergency panproctocolectomy with permanent end ileostomy

E) Oral azathioprine

A) Topical aminosalicylates

•Topical aminosalicylates are indicated for managing mild or moderate proctitis and proctosigmoiditis.

B) High-dose oral aminosalicylates

•High-dose oral aminosalicylates are indicated in the management of left-sided or extensive mild to moderate ulcerative colitis.

C) Intravenous corticosteroids

•According to Truelove and Witt's severity score, this exacerbation of ulcerative colitis is classified as severe. Hence, intravenous corticosteroids should be considered as first-line management.

D) Emergency panproctocolectomy with permanent end ileostomy

•Emergency surgery such as panproctocolectomy with permanent end ileostomy is considered in those with acute fulminant ulcerative colitis and worsening symptoms despite intravenous corticosteroid use.

E) Oral azathioprine

•Oral azathioprine is used as the first-line treatment to maintain remission of Crohn's disease.

Truelove and Witt's criteria for severity of UC flare

	Mild	Moderate	Severe
Bowel movements (no. per day)	Fewer than 4	4-6	6 or more plus at least one of the features of systemic upset (marked with * below)
Blood in stools	No more than small amounts of blood	Between mild and severe	Visible blood
Pyrexia (temperature greater than 37.8°C)	No	No	Yes
Pulse rate greater than 90 bpm	No	No	Yes
Anaemia (< 10g/100mL)	No	No	Yes
Erythrocyte sedimentation rate (mm/hour)	30 or below	30 or below	above 30

UC management

Mild-moderate disease

If step 1 does not work after 4 weeks, or symptoms worsen, move to step 2.

Moderate first presentation:
Step 1: topical ASA
Step 2: oral ASA

Proctitis and proctosigmoiditis:
Step 1: Topical ASA or oral ASA.
Step 2: Consider adding oral prednisolone. If this does not help after 2-4 weeks or symptoms worsen, consider adding oral tacrolimus.

Left sided or extensive disease
Step 1: High dose oral ASA.
Step 2: Consider adding oral prednisolone. If this does not help after 2-4 weeks or symptoms worsen, consider adding oral tacrolimus.

Acute severe disease

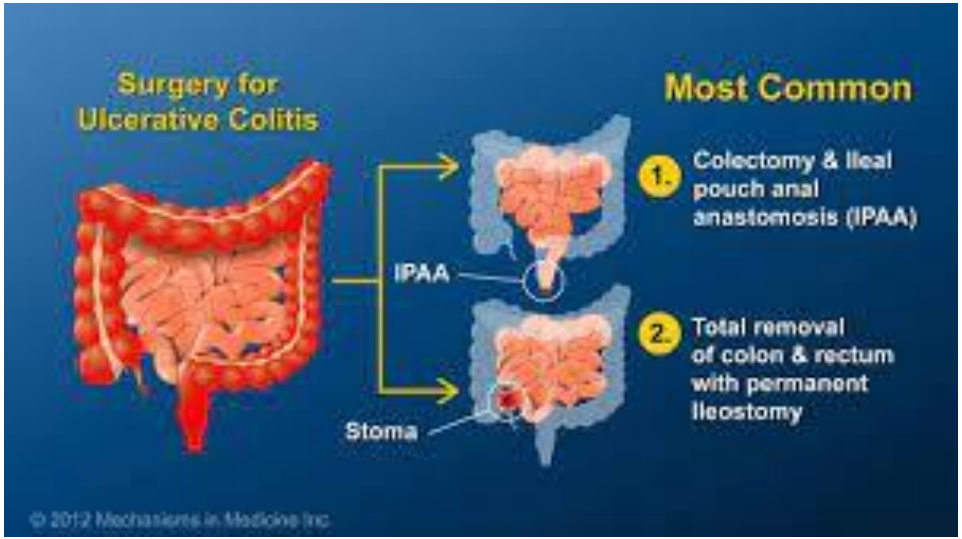
Step 1: IV corticosteroids (if contraindicated or not tolerated, use IV ciclosporin).

Step 2: If no improvement in 72 hours or worsening symptoms, add IV ciclosporin or consider surgery (if IV ciclosporin contraindicated or not tolerated, consider infliximab).

UC surgical options

Acute indication: symptoms worsening despite intravenous steroids

Elective indication: when there is failure to induce remission by medical means



	Crohn's disease	Ulcerative colitis
Location	Whole gastrointestinal tract	Colon (mainly rectum)
Type of lesion	Transmural inflammation	Superficial ulcers
Characteristic pattern	Skip lesions	Continuous lesions
Microscopic findings	Non-caseating granulomas	Crypt abscesses
Macroscopic findings	Cobblestone appearance	Friable mucosa
Complications	•Strictures •Bowel obstruction •Fistulas •Perianal abscesses	•Gastrointestinal bleeding •Toxic megacolon •Peritonitis •Adenocarcinoma
Treatment options	•Immunosuppressants (e.g. corticosteroids) •Aminosalicylates (e.g. mesalazine) •Antibiotics (e.g. ciprofloxacin and metronidazole) •Biologics (e.g. infliximab) •**Immunomodulators (e.g. azathioprine)** •Surgery (not curative)	•Immunosuppressants (e.g. corticosteroids) •Aminosalicylates (e.g. mesalazine) •Biologics (e.g. infliximab) •Surgery (curative)

SBA

A medical student attends an occupational health appointment before starting her clinical placements. She moved to the UK 4 years ago. She reports no symptoms and is eager to begin her placement. A routine blood test for hepatitis B serology is performed which shows:

HBsAg: Positive
IgG anti-HBc antibody: Positive
IgM anti-HBc antibody: Negative
anti-HBs antibody: Negative
HBeAg: Negative
anti-HBe-antibody: Positive

Which of the following is the correct interpretation of her hepatitis B serological testing?

A) Chronic hepatitis B infection with high infectivity
B) Acute hepatitis B infection
C) Chronic hepatitis B infection with low infectivity
D) Immunity to hepatitis B due to natural infection
E) Immunity to hepatitis B due to vaccination

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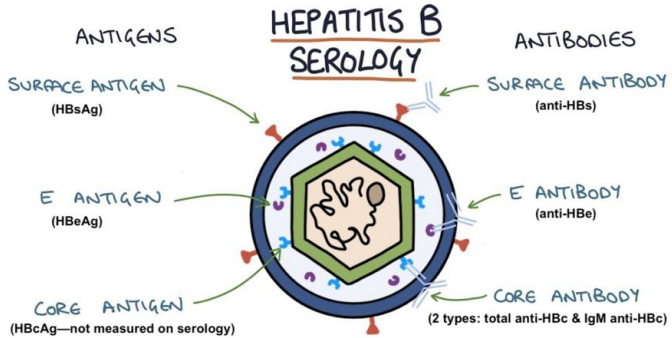

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Hepatitis B serology interpretation

Status/Marker	HBsAg	anti-HBs	IgM anti-HBc	IgG anti-HBc	HBeAg	anti-HBe	HBV DNA
Acute HBV Infection with Immunity	Positive	Negative	Positive	Positive	Positive	Negative	Positive
Chronic HBV Infection	Positive	Negative	Negative	Positive	Positive/Negative	Positive/Negative	Positive/Negative
Immune due to Natural Infection	Negative	Positive	Negative	Positive	Negative	Positive	Negative
Immune due to Hepatitis B Vaccination	Negative	Positive	Negative	Negative	Negative	Negative	Negative

	Hepatitis A	Hepatitis B	Hepatitis C	Hepatitis D	Hepatitis E
Spread	-faecal-oral route -contaminated food -anal sex	Infected blood, bodily fluids, sex, transfusion, vertical	Infected blood, bodily fluids, sex, transfusion, vertical	Bodily fluids, blood, sex	- Faecal-oral
Incubation period	2-6 weeks	2-3 months	6-9 weeks	Hepatitis D only occurs as a superinfection in patients with concurrent hepatitis B infection	Average 5-6 weeks
Chronicity	Can take up to 6 months to clear infection	HBsAg is detected 3-5 weeks after infection. If present for >6 months, this defines carrier status (5-10%)	Yes	Yes	In immunosuppressed
Management	Supportive	Peginterferon alfa-2a is the first line, with tenofovir and entecavir as second-line alternatives.	Antivirals	Antiviral	Supportive
Vaccine	Yes	Yes	No	(Hep B vaccine can prevent)	No

SBA

A 28-year-old lady comes into the Emergency Department having ingested 25 tablets of paracetamol about 3 hours ago. She is currently asymptomatic. Her observations are as follows: HR 100, RR 18, BP 130/80, T 37.0, SO2 99% RA.

According to local protocol, she would have to wait an hour and have her paracetamol levels tested at 4 hours before starting treatment. However, the Emergency Department consultant wants to start her on NAC treatment now.

Which of the following factors would likely be the reason for the consultant's decision?

A) History of anorexia nervosa
B) Family history of hepatocellular carcinoma
C) All 25 tablets were taken at the same time
D) Previous paracetamol overdose
E) Use of oral contraceptive pill

History of anorexia nervosa

- There is increased risk of paracetamol toxicity in patients that are in glutathione deplete states. This includes eating disorders, HIV and malnutrition. A history of anorexia nervosa would warrant immediate administration of NAC

Family history of hepatocellular carcinoma

- This patient should be started on NAC treatment immediately if she has certain conditions that increases her risk of toxicity. Pre-existing liver disease would put her at an increased risk, but having a family history of HCC does not mean that she has pre-existing liver disease

All 25 tablets were taken at the same time

- Only in cases of a staggered overdose, where different amount of tablets were taken over a long period of time, should NAC be administered immediately

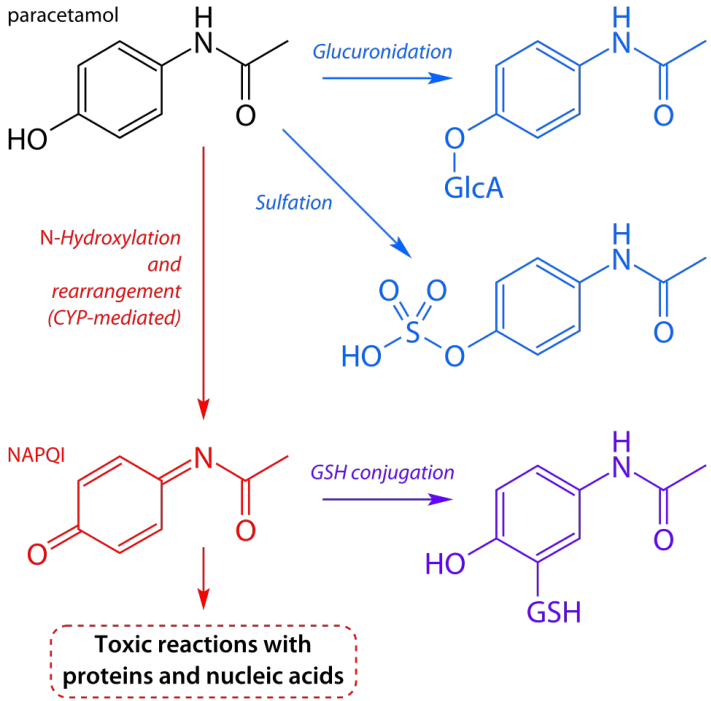
Previous paracetamol overdose

- This patient should be started on NAC treatment immediately if she has certain conditions that increases her risk of toxicity. Previous paracetamol overdose does not increase the risk of toxicity

Use of oral contraceptive pill

- There is an increased risk of toxicity if patients are on long term medications that are enzyme inducers. OCPs do not induce liver enzymes

Paracetamol metabolism

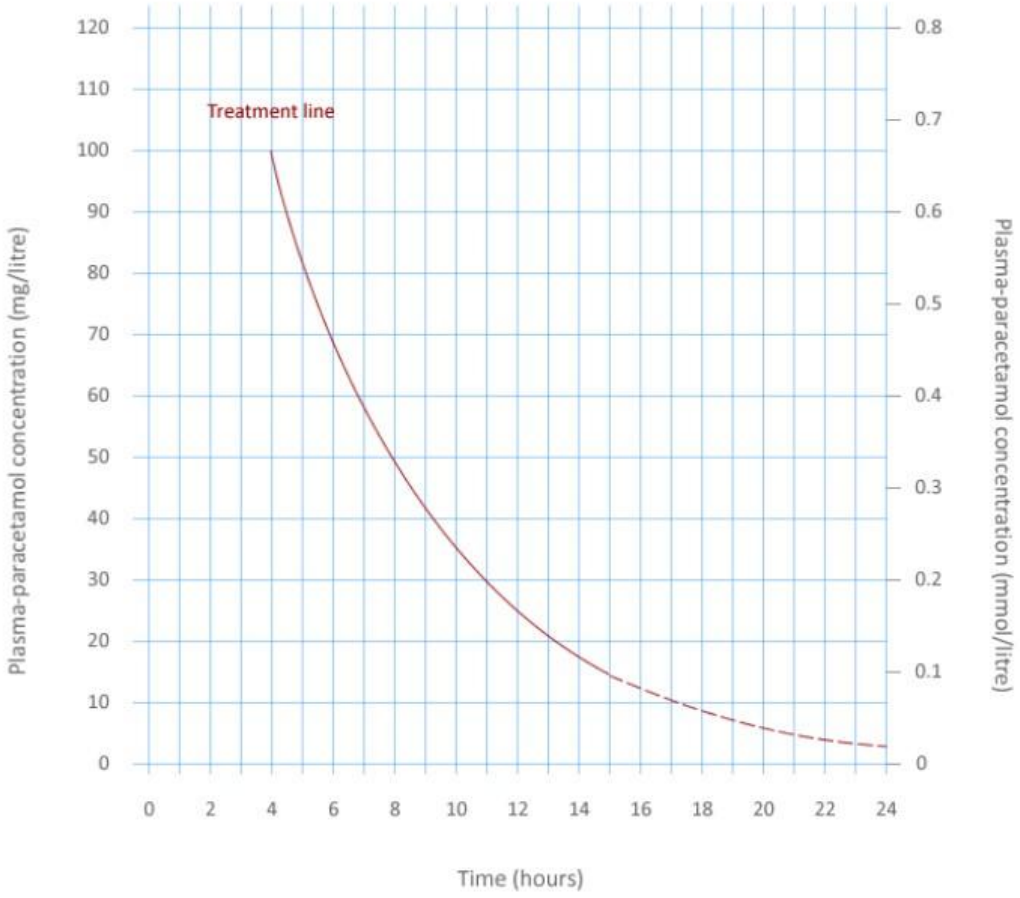


Risk factors

- History of self-harm
- History of frequent/repeated use of pain relief medication
- Low body weight (<50kg)
- Cytochrome P450 inducers (e.g. phenytoin, phenobarbital, rifampicin)
- Glutathione deficiency (malnourishment e.g. eating disorders, alcohol misuse, chronic illness affecting nutritional intake)

Management

Overdose category	Assessment & management
Acute overdose (ingested over one hour or less)	
Ingestion less than 1 hour ago + dose >150mg/kg:	•Administer activated charcoal
Presenting <4 hours after last ingestion	• Wait until 4 hours to take a level and treat with N-acetylcysteine based on level
Presenting 4-8 hours after last ingestion	•Take blood samples immediately •Start N-acetylcysteine immediately if there is going to be a delay of ≥8 hours in obtaining the paracetamol level •Start acetylcysteine if **4-hour paracetamol level** is above the treatment line, or evidence of liver injury (usually raised ALT, jaundice, RUQ pain, INR>1.3)
Presenting 8-24 hours after last ingestion	•Take blood samples immediately •**If ≥150mg/kg ingested (or unknown amount) or symptomatic**: start acetylcysteine while waiting for results •**If <150mg/kg ingested**: wait for results, start acetylcysteine if paracetamol level above the treatment line, or evidence of liver injury
Presenting more than 24 hours after last ingestion	•Take blood samples immediately •**If ≥150mg/kg ingested (or unknown amount) or symptomatic**: start acetylcysteine while waiting for results •**If <150mg/kg ingested**: wait for results, start acetylcysteine if ALT raised, INR >1.3 (in the absence of other cause) or paracetamol detected
Staggered paracetamol overdose (ingested over >1 hour)	
All patients	•Start acetylcysteine treatment **immediately** •Take blood samples four hours after the last ingestion •Consider discontinuing acetylcysteine if low risk of hepatotoxicity: paracetamol concentration <10mg/L, normal ALT, INR <1.3 and asymptomatic
Therapeutic excess	
All patients	•**If symptomatic**: start acetylcysteine treatment immediately •Management of therapeutic excess depends on the patient's weight and quantity of paracetamol consumed within the last 24 hours


Treatment line
Plasma-paracetamol concentration (mg/litre)
Plasma-paracetamol concentration (mmol/litre)
Time (hours)
120
110
100
90
80
70
60
50
40
30
20
10
0
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
2
4
6
8
10
12
14
16
18
20
22
24

King's College Criteria

Parameter	Criteria
Arterial pH	Less than 7.3, irrespective of the grade of encephalopathy
Serum creatinine	Greater than 3.4 mg/dL (300 µmol/L), irrespective of the grade of encephalopathy
Prothrombin time	Greater than 100 seconds
Grade III or IV encephalopathy	Plus either bilirubin greater than 18 mg/dL (300 µmol/L) or international normalized ratio (INR) greater than 6.5

SBA

A 47-year-old woman presents to her GP complaining of epigastric discomfort which has been gradually worsening over four months. The pain is worst upon waking and mid-morning and is relieved by eating. There is no history of weight loss, melaena or dysphagia. On examination, there is slight epigastric tenderness but no other findings of note. Which of the following is the most likely diagnosis?

A) Gastritis
B) Gastro-oesophageal reflux disease
C) Gastric adenocarcinoma
D) Duodenal ulcer
E) Gastric ulcer

SBA

She has a past medical history of severe asthma, controlled by daily oral prednisolone, montelukast and a combined steroid inhaler. She also reports taking paracetamol for a sprained ankle and taking the progestogen-only pill.

Which medication is the most likely cause of the underlying pathology?

A) Paracetamol
B) Oral prednisolone
C) Salbutamol inhaler as required
D) Montelukast
E) Progesterone-only pill

Gastritis

- Inflammation of the stomach can also cause epigastric pain, however, the pain is typically worse after meals

Gastro-oesophageal reflux disease

- GORD can also cause epigastric abdominal pain, however, the pain is typically after meals and is worsened when lying flat. The pain is often more retrosternal and may be described by the patient as heartburn

Gastric adenocarcinoma

- The lack of weight loss, the association of the pain to before mealtimes, and the lack of other more sinister red flag symptoms points away from this diagnosis, however it would be an important differential to rule out

Duodenal ulcer

- Duodenal ulcers are four times as common as gastric ulcers and the pain is typically worse before food or at night and is relieved by food or milk. Epigastric tenderness is a common examination finding. There are no red flag symptoms, such as weight loss. However, weight loss can occur with ulcers, especially gastric ulcers

Gastric ulcer

- Gastric ulcers are less common compared with duodenal ulcers. The epigastric pain is typically worsened by eating. Weight loss is more likely to be associated with gastric ulcers compared with duodenal ulcers

SBA

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Which medication is the most likely cause of the underlying pathology?

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D) Montelukast
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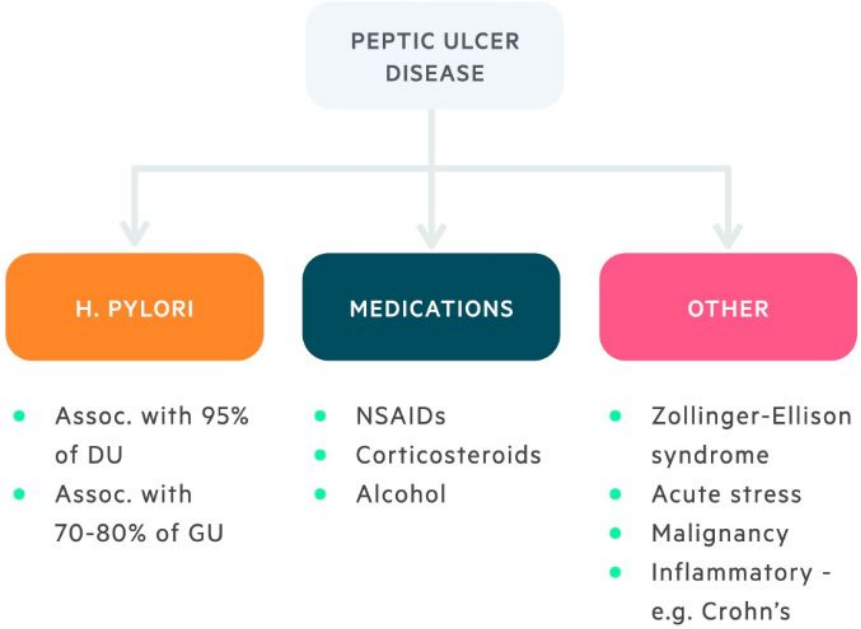
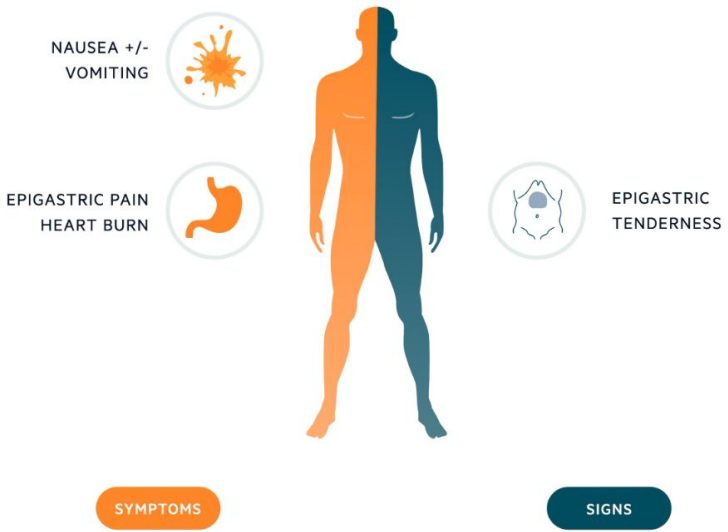
Peptic ulcer disease





Investigations

- Patients aged >55 with weight loss and dyspepsia should be referred for an urgent OGD to investigate for oesophageal and gastric cancer

- Offer the carbon-13 urea breath test first-line, or the stool antigen test second-line if the carbon-13 urea breath test is not available — ensure the person has not taken a PPI in the past 2 weeks, or antibiotics in the past 4 weeks.

SBA

A 39-year-old man attends the endoscopy department for a routine oesophagogastroduodenoscopy after experiencing dyspepsia and epigastric pain for the past 7 weeks. He is found to have a gastric ulcer and a 13C-urea breath test is performed, which is positive for *Helicobacter pylori*. He informs you that his GP recently gave him co-amoxiclav and he developed tongue swelling, difficulty breathing and a widespread rash.

Which of the following treatment options is most suitable for this patient?

A) Omeprazole, clarithromycin and metronidazole for 7 days
B) Omeprazole for 7 days
C) Omeprazole, clarithromycin and metronidazole for 3 days
D) Omeprazole, amoxicillin and metronidazole for 7 days
E) Omeprazole, amoxicillin and clarithromycin for 7 days

Management

- Management of H. Pylori-negative peptic ulcer disease involves a 4–8-week course of full-dose PPI treatment in conjunction with lifestyle advice.

- If the patient is H.pylori positive with a proven gastric/duodenal ulcer which is:
  - Associated with NSAID use: 8-week PPI therapy followed by first-line eradication therapy
  - Not associated with NSAID use: eradication therapy with PPI

For patients with gastric ulcers, a repeat endoscopy 6-8 weeks after the start of PPI treatment is recommended to ensure ulcer healing and rule out malignancy, as well as H.pylori re-testing (C-13 urea breath test first-line, stool antigen test second line) if appropriate.

Re-test for h.pylori if continue to have symptoms despite first line eradication therapy

H.Pylori eradication

First line:
A PPI twice-daily and amoxicillin 1 g twice-daily and

- Either clarithromycin 500 mg twice-daily or metronidazole 400 mg twice-daily.

If the person is allergic to penicillin, offer a 7-day triple therapy regimen of:
A PPI twice-daily and clarithromycin 500 mg twice-daily and metronidazole 400 mg twice-daily.

If the person is allergic to penicillin and has had previous exposure to clarithromycin, offer a 7-day quadruple therapy regimen of:
A PPI twice-daily and metronidazole 400 mg twice-daily and tetracycline hydrochloride 500 mg four times daily and bismuth subsalicylate 525 mg four times-daily.

Second line (if a person has ongoing symptoms following first-line eradication and H. pylori re-testing is positive)

Offer a 7-day triple therapy regimen of:
A PPI twice-daily and amoxicillin 1 g twice-daily and
Either clarithromycin 500 mg twice-daily or metronidazole twice-daily 400 mg (whichever was not used first-line).

If the person has had previous exposure to clarithromycin and metronidazole, offer a 7-day triple therapy regimen of:
A PPI twice-daily and amoxicillin 1 g twice-daily and
Either levofloxacin 250 mg twice-daily or tetracycline hydrochloride 500 mg four times a day.

If the person is allergic to penicillin and has not had previous exposure to levofloxacin, offer a 7- day triple therapy regimen of:
A PPI twice-daily and metronidazole 400 mg twice-daily and levofloxacin 250 mg twice-daily.

If the person is allergic to penicillin and has had previous exposure to levofloxacin, offer a 7-day quadruple therapy regimen of:
A PPI twice-daily and bismuth subsalicylate 525 mg four times a day and metronidazole 400 mg twice-daily and tetracycline hydrochloride 500 mg four times a day.

Complications

- Perforation
- Upper GI bleed
- Gastric outlet syndrome

SBA?

Which of the following describes the pathophysiology of pre-hepatic jaundice?

A) Obstruction in the biliary tree
B) Autoimmune anti-neutrophil cytoplasmic antibodies (ANCA) attack the bile ducts
C) Excessive red blood cell breakdown
D) Immature activation of precursor enzymes
E) Hepatocellular damage

Obstruction in the biliary tree

•This is not what causes pre-hepatic jaundice. Post-hepatic jaundice is caused by obstruction of the biliary tree and impaired drainage means the bilirubin that is not excreted from the body will have been conjugated by the liver but its passage to the gastrointestinal (GI) tract via the biliary tree is blocked. Hence the result is a conjugated bilirubinaemia

Autoimmune anti-neutrophil cytoplasmic antibodies (ANCA) attack the bile ducts

•This is not what causes pre-hepatic jaundice. Autoimmune attack of the bile ducts by ANCA describes primary sclerosing cholangitis which leads to scarred and inflamed bile ducts. This is a cause of post-hepatic jaundice

Excessive red blood cell breakdown

•In pre-hepatic jaundice, excessive red blood cell breakdown overwhelms the liver's ability to conjugate bilirubin. This causes an unconjugated bilirubinaemia where any bilirubin that manages to become conjugated will be excreted normally, yet it is the unconjugated bilirubin that remains in the bloodstream to cause the jaundice.

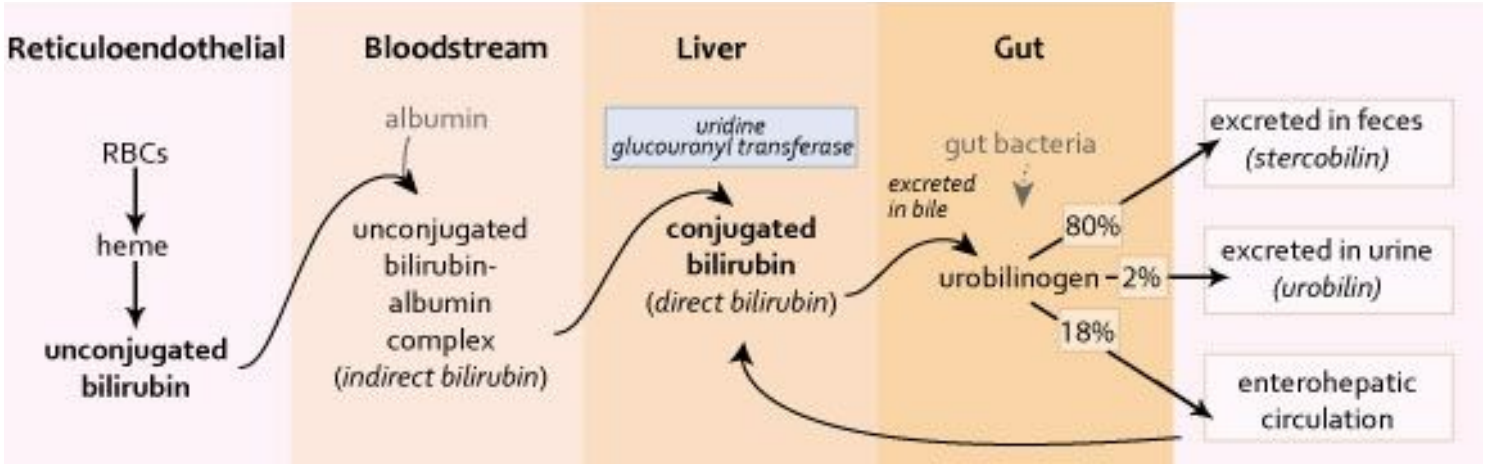
Immature activation of precursor enzymes

•This is not what causes pre-hepatic jaundice. This describes the pathophysiology of acute pancreatitis. Under extreme disruption of pancreatic function, the pancreas begins to autolyse and release pancreatic enzymes including amylase and lipase

Hepatocellular damage

•This is not what causes pre-hepatic jaundice. Hepatic jaundice is caused by dysfunction of the hepatic cells. The liver loses its ability to conjugate bilirubin. In cases where the liver becomes cirrhotic, it compresses the intra-hepatic portions of the biliary tree to cause a degree of obstruction. This leads to both unconjugated and conjugated bilirubin in the blood

Bilirubin metabolism



https://step1.medbullets.com/gastrointestinal/110032/bilirubin-production-and-excretion

Pre-hepatic jaundice

Any cause of increased bilirubin production:

- Haemolytic disorders (sickle cell disease, hereditary spherocytosis, autoimmune haemolytic anaemia)
- Iatrogenic (e.g. metallic heart valve)

Features of pre-hepatic jaundice:

- Anaemia (due to haemolysis)
- Unconjugated hyperbilirubinaemia (due to excess bilirubin that has not yet been conjugated in the liver)
- Markers of haemolysis (raised LDH, raised reticulocytes, low haptoglobin)

Hepatic jaundice

Unconjugated hyperbilirubinaemia (abnormal bilirubin processing)	Inherited causes: Gilbert's syndrome, Crigler-Najjar syndrome
	Acquired causes: Hyperthyroidism, Ethinyl estradiol, antibiotics (e.g. gentamicin at high serum levels), anti-retroviral drugs, others
Conjugated hyperbilirubinaemia (abnormal bilirubin excretion)	Hepatocellular injury: viral hepatitis, alcohol, fatty liver, autoimmune hepatitis, hereditary haemochromatosis, Wilson's disease
	Drugs, including paracetamol overdose, halothane, valproate, statins, tuberculosis antibiotics
	Infiltrative disorders: sarcoidosis, amyloidosis, TB
	Malignancy
	Inherited disorders (rotor and Dubin-Johnson syndromes)

SBA

Which of the following would you expect to be elevated in post-hepatic jaundice?

A) Alkaline phosphatase
B) Iron
C) Mean cell volume (MCV)
D) Creatine kinase
E) Unconjugated bilirubin

Alkaline phosphatase

- In post-hepatic jaundice, ALP and gamma-glutamyl transferase (GGT) are raised. This is termed a "cholestatic" picture and indicates there is an obstruction in the biliary tree that is causing post-hepatic jaundice. This obstruction can be due to gallstones, external compression from other organs or scarring of the bile ducts

Iron

- This would not be raised in post-hepatic jaundice. Raised iron levels can point towards haemochromatosis. This disease can cause hepatocellular damage and cirrhosis which would produce hepatic jaundice, not post-hepatic jaundice

Mean cell volume (MCV)

- This would not be raised in post-hepatic jaundice. A raised MCV alongside low haemoglobin levels indicates macrocytic anaemia of which vitamin B12 deficiency is a common cause. Pernicious anaemia is the most common cause of vitamin B12 deficiency which causes pre-hepatic jaundice, not post-hepatic jaundice

Creatine kinase

- This would not be raised in post-hepatic jaundice. Creatine kinase is raised in muscle diseases such as muscular dystrophy and rhabdomyolysis. This indicates acute kidney injury, not post-hepatic jaundice

Unconjugated bilirubin

- Unconjugated bilirubin would not be raised in post-hepatic jaundice but conjugated bilirubin would be. This is because the liver is functioning normally and retains its ability to conjugate bilirubin, therefore there would not be a high level of unconjugated bilirubin. Conjugated bilirubin is being blocked from passing through the biliary tree to the duodenum which causes it to seep into circulation

Liver enzymes and jaundice

Hepatic enzymes include ALT, AST, gGT, and ALP

- Pre-hepatic jaundice: typically, normal enzymes with an isolated elevation in unconjugated bilirubin
- Hepatic jaundice (conjugated - i.e. liver parenchyma): usually a predominant rise in ALT/AST > ALP/gGT
- Post-hepatic jaundice (i.e. biliary problem): usually a predominant rise in gGT/ALP > ALT/AST

Post-hepatic jaundice

Intrahepatic vs extrahepatic cholestasis

Characterised by:

- Elevated conjugated bilirubin
- Cholestatic liver enzyme elevation (e.g. predominant rise in ALP and GGT)
- Evidence of obstruction on imaging

Some causes:

- Autoimmune (e.g. primary biliary cirrhosis, primary sclerosing cholangitis)
- Gallstones
- Mirizzi's syndrome: common hepatic duct obstruction caused by extrinsic compression from an impacted stone in the cystic duct or infundibulum of the gallbladder
- Drugs including coamoxiclav, flucloxacillin, nitrofurantoin, steroids, sulfonylureas
- Malignancy (e.g. head of pancreas, cholangiocarcinoma)
- Biliary atresia

SBA

A 23-year-old man presents to his GP after noticing that his skin was becoming slightly discoloured and his sclera were more yellow. He reports feeling otherwise well but has been a bit more stressed recently because of exams. He had a similar episode to this during his exams last year. He is eating less than usual owing to the stress, but he still has a normal appetite. On examination, he has scleral icterus and no abdominal tenderness. Which of the following investigation findings is most in keeping with the likely diagnosis?

A) Alkaline phosphatase (ALP) 231 U/L (raised)
B) Unconjugated bilirubin 54 micromol/L (raised)
C) Conjugated bilirubin 63 micromol/L (raised)
D) Bilirubin present on urine dip
E) Alanine aminotransferase (ALT) 579 U/L (raised)

Alkaline phosphatase (ALP) 231 U/L (raised)

•`These is no cholestasis in Gilbert syndrome, so the ALP will be normal.

Unconjugated bilirubin 54 micromol/L (raised)

•This is a typical history of Gilbert syndrome. It is a benign condition where patients experience intermittent mild jaundice in relation to stress, fasting, infection or exercise. The key manifestations in the blood tests are a slightly elevated bilirubin. As Gilbert syndrome is due to a decreased activity of the enzyme that conjugates bilirubin, it causes a mild unconjugated hyperbilirubinaemia.

Conjugated bilirubin 63 micromol/L (raised)

•Although the bilirubin is mildly elevated in Gilbert syndrome, it is an unconjugated hyperbilirubinaemia owing to reduced activity of the enzyme that conjugates bilirubin.

Bilirubin present on urine dip

•Unconjugated bilirubin is not water-soluble so would not appear in the urine (unlike conjugated bilirubin).

Alanine aminotransferase (ALT) 579 U/L (raised)

•The transaminases in Gilbert syndrome are normal as there is no hepatocellular damage.

Questions?

Summary: gastroenteritis + hepatitis viruses

- Gastroenteritis is a prevalent condition, often leading to diarrhoea and vomiting. It is typically caused by the ingestion of bacteria, viruses, or toxins. Key signs and symptoms include diarrhoea, vomiting, and occasionally, acute kidney injury in frail individuals. The primary management strategy is conservative with fluid replacement or oral rehydration sachets. Antibiotics may be used in severe cases, particularly in patients who are systemically unwell, immunosuppressed, or elderly.

- Hepatitis means inflammation of the liver and may be caused by a range of infectious and non-infectious aetiologies. All infectious hepatitis cases are notifiable diseases in the UK. The most common causes of viral hepatitis in the UK are hepatitis A, B and C viruses. These can all cause acute disease, but HBV and HCV can also cause chronic infection. The latter can lead to liver fibrosis and hepatocellular carcinoma.

Summary IBD

- Crohn's disease (CD) is a chronic, relapsing inflammatory bowel disease (IBD) characterized by transmural granulomatous inflammation. Key signs and symptoms include gastrointestinal and systemic symptoms, such as crampy abdominal pain, diarrhoea, weight loss, and fever. The disease is diagnosed primarily through blood tests and endoscopy with imaging. Management strategies include monotherapy with glucocorticoids, azathioprine, mercaptopurine, and biological agents for severe cases. Surgical management is rarely curative and should be maximally conservative.

- Ulcerative colitis (UC) is a chronic inflammatory disease that affects the large bowel. Symptoms include diarrhea, urgency, tenesmus, weight loss, and fever. The disease can present with extra-intestinal features such as dermatological, ocular, musculoskeletal, and hepatobiliary manifestations. Investigations include blood tests, microbiological investigations, endoscopic investigations, and imaging. The management of UC is based on severity and involves inducing and maintaining remission using medications like aminosalicylates, steroids, and biologics. Surgery may be required in severe cases or when medical treatment is unsuccessful. Long-term complications include colorectal cancer, cholangiocarcinoma, colonic strictures, and Primary Sclerosing Cholangitis.

Summary: paracetamol overdose + peptic ulcer disease

- Paracetamol overdose, the excessive use of paracetamol, accounts for 44% of all adult self-poisoning cases in the UK and results in approximately 150,000 hospital admissions annually. Key signs and symptoms can range from no symptoms to nausea, vomiting, loin pain, haematuria, proteinuria, jaundice, abdominal pain, coma, and severe metabolic acidosis. Investigations should include blood tests such as Full Blood Count, Liver Function, Urea and Electrolytes, Clotting, and Venous Blood Gas. Management depends on the timing of ingestion and the patient's clinical condition, with N-acetylcysteine being the mainstay of treatment. The decision to treat is guided by a nomogram, although in certain situations, N-acetylcysteine should be started immediately.

- Peptic ulcer disease, encompassing both duodenal and gastric ulcers, is a condition where the stomach lining's self-protection mechanisms fail, often due to the presence of external factors like H. Pylori. Key signs and symptoms include pain, nausea, vomiting and loss of appetite. The primary investigation tool is endoscopy, which allows for a direct visual inspection of the ulcers. Management strategies include both pharmaceutical interventions, such as PPI treatment, and lifestyle changes, like cessation of smoking and dietary adjustments.

Summary: jaundice

- Bilirubin is a breakdown product of red blood cell degradation. The presence of bilirubin in the urine may be an early indicator of liver disease. Bilirubin is conjugated with glucuronic acid by glucuronyltransferase and can then be excreted in the bile. In the bowel, bilirubin is converted to stercobilin by gut flora, which is then excreted in the faeces, as well as urobilinogen, which is reabsorbed and converted into bile, excreted in the faeces or excreted in the urine. Jaundice can be divided into pre-hepatic, hepatic and post-hepatic causes.

To summarise

- Medicine is hard
- But you can do it

- Quesmed
- Pulsenotes
- Geeky Medics
- NICE
- BMJ Best Practice

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