Griffith University Medicine Society
Gold Coast, Australia

# Year 1 Peer Based Learning 2018

# GI System

Please note – this learning resource has been produced by the GUMS Academic Team. It is possible that there are some minor errors in the questions/answers, and other possible answers that are not included below. Make sure to check with other resources.

## Case 1

Patient has a history of rheumatoid arthritis for which he takes NSAIDs. Presents complaining of epigastric pain and non-bilious, coffee-ground vomitus.

**What is the significance of the vomit being non-bilious and coffee ground?**

- *Coffee ground = blood that has been digested (eg, from stomach or further down, not from oesophagus)*
- *Non-bilious = does not contain bile = bleeding from before the duodenum*

**Explain the pathophysiology of the most likely cause of epigastric pain & vomiting in THIS patient.**

*NSAIDs have a few effects on the stomach:*

- *LESS MUCOUS (inhibit COX enzymes → decreased prostaglandin production → less mucous & HCO3 production = less protection of mucosa)*
- *MORE HCL (inhibit COX enzymes → decreased PG production → less inhibition of HCl secretion = more acid = more irritation)*
- *Direct chemical irritation to mucosa*

*Irritation to the mucosa leads to pain and bleeding. Bleeding (and possibly pain if severe enough) stimulates may stimulate vomiting.*

**What are other risk factors for this condition (not necessarily in this patient)? Explain how they contribute to the pathogenesis of the disease.**

- *H pylori – invades mucosa*
    - *Produces toxins & inflammatory mediators*
    - *Has urease enzyme (converts urea to ammonia to increase pH, to protect bacteria from H+). Increased pH leads to increased acid production, causing further damage to the mucosa*

- *Smoking (impairs blood flow, reduces healing of damaged gastric mucosa)*

**Imagine the patient was asked to do a urea breath test. Explain the basis of this test.**

*Aims to detect H pylori bacteria.*

- *Swallow a capsule containing urea labelled with an uncommon isotope. Wait ~30 minutes.*
- *If H pylori (contains urease enzyme) is present, it breaks urea into carbon dioxide & ammonia. The CO2 will be labelled with the isotope and will be expelled and detected in the breath.*
- *If no CO2 labelled with the isotope is present – no H pylori*

**How should THIS patient be treated (and explain the basis of treatment)? What about if a urea breath test was positive?**

*This patient:*

- *Stop NSAID (if possible)*
- *PPI – proton pump inhibitor, to reduce HCl production*

*If urea breath test was possible – indicates H pylori resistance, so use triple-therapy:*

- *PPI*
- *Amoxicillin – antibacterial*
- *Clarithromycin – antibacterial*

*Review mechanisms of action of antibiotics.*

**If this progressed without treatment, what possible consequences might you see and how would you detect these/how would they present?**

- *Perforation → pneumoperitoneum*
  - *Detected on erect X-ray (air under the diaphragm – air rises)*
  - *Pain (abdominal pain, think about patterns of referred pain in this patient)*
  - *Could lead to peritonitis*
  -
- *Bleeding → iron deficiency anaemia, haemorrhage*

Ruptured gastric ulcer on the lesser curvature of stomach → bleeding from left gastric artery.
An ulcer on the posterior wall of duodenum → bleeding from gastroduodenal artery.

**Imagine this patient has a gastrectomy. Thinking about the functions of the stomach, what advice would you give this patient (and why)?**

- *Have smaller, more frequent meals. Otherwise the small intestine will be overwhelmed and food will remain undigested in the intestines, causing osmotic diarrhoea (Dumping syndrome)*

**What would you consider prescribing a patient following a gastrectomy and why? If you don't, what might happen?**

- *Vitamin B12 injections. Lack of intrinsic factor secreted by stomach. Intrinsic factor is required for Vitamin B12 absorption (by binding to it).*
- *A Vitamin B12 deficiency can lead to pernicious anaemia.*

**Is anything absorbed or chemically digested in the stomach?**

- *Yes – some aspirin and alcohol are absorbed*
- *Yes – proteins can be digested by pepsin*

**Briefly explain the impact of the following drugs on the gastric acid secretion. Think about the specific receptors and cellular processes involved.**

- **Proton pump inhibitor** *Decrease HCl production. This blocks the H+/K+ ATPase pump, decreasing the efflux of H+ into the stomach*

- **H1 antagonist** *No change! There are no H1 receptors in the stomach. H2 receptors are found in the stomach. A H2 antagonist would decrease gastric acid production.*
- **Muscarinic agonist** *Increase HCl production. This mimics the effects of the parasympathetic nervous system.*

**What if: This patient started taking an ACE-inhibitor and loop diuretic for hypertension and heart failure. What condition do you have to closely monitor for? Explain the pathophysiological basis behind this.**

Prostaglandins dilate afferent arteriole; angiotensin II constricts efferent. Loop diuretic causes fluid loss → all lower GFR – this is known as the 'triple whammy'
PDA ACE

## Case 2

A patient presents complaining of passing fatty, bulky stools with an offensive odour. She states that they are difficult to flush in the toilet.

**What is this sign called?**

- Steatorrhea

**There are many possible causes of this. Based on the additional symptoms described in the table below, think about a possible diagnosis, and explain the cause of fatty, bulky, malodorous stools.**

| Additional Symptoms | Diagnosis | Cause of fatty, bulky, malodorous stools |
|---|---|---|
| **Recent onset of epigastric pain radiating to the back, tenderness on abdominal palpation, nausea, vomiting** | *Acute pancreatitis* | *Lack of lipase → unable to break triglycerides (to fatty acids and glycerol) → less fat absorption from intestine → more fat remains in intestine → steatorrhea* |
| **Treated with broad-spectrum antibiotics in hospital for the last 2 weeks**<br>**Bonus question: what is the most common bug that causes infection in hospitals following antibiotic use?** | *Bacterial overgrowth*<br><br>*Clostridium difficile* | *Antibiotic use → kills normal gut flora → overgrowth of pathogenic bacteria → less bile acid deconjugation/ enterohepatic recycling → bile acids remain in GIT → steatorrhea* |
| **Greatly elevated serum ALP and GGT, painless jaundice** | *Bile duct obstruction/ cholestasis, due to pancreatic head tumour*<br><br>**(explain that PAINLESS jaundice suggests distal obstruction of bile duct as oppose to something like cholelithiasis)** | *Lack of bile salts entering duodenum → less fat emulsification → less fat absorption → steatorrhoea* |
| **Inflammatory changes in small intestinal mucosa, positive anti-endomysium and anti-tissue transglutaminase antibodies** | *Coeliac disease* | *Mucosal lesions → loss of enterocytes → less absorption → steatorrhoea* |
| **Recent surgical resection of ileum due to adenocarcinoma** | | *No ileum (where bile salts are usually reabsorbed) → lack of bile salt reabsorption → steatorrhoea* |

**This should prompt discussion of these diseases e.g. histological findings in coeliac disease, key features of Crohn's and ulcerative colitis etc.**

CASE 1: GUT STUFF AND BUTT STUFF

A patient presents to you in the community with complaints of recurrent abdominal pain.

**If the pain localised to RLQ, what would your differentials be (female)?**

- Appendicitis
- Ectopic pregnancy
- Diverticulitis

**If the pain localised to the RUQ, what would your differentials be?**

- Liver pathology (ex. cirrhosis, fatty liver, etc.)
- Gall bladder/biliary tree pathology (ex. cholecystitis, cholangitis, etc.)
- Others: duodenum, pancreas, colon, kidney, diaphragm

**If the patient's stool is positive for blood, what would your differentials be?**

- Ulcerative colitis
- Colorectal cancer
- Anal fissures/hemorrhoids
- Any infectious causes
- Any ischemic causes

**Inflammatory bowel disease is often seen in young women as recurrent bouts of bloody diarrhea and abdominal pain, compare its subclassifications: 1) ulcerative colitis and 2) Crohn disease.**

| | **Ulcerative colitis** | **Crohn disease** |
|---|---|---|
| Wall involvement | Mucosal/submucosal ulcers | *Full thickness* ulcers |
| Location | Beings in rectum and extends from there *continuously* | Anywhere in GI tract, contains *skip lesions* (terminal ileum most common) |
| Gross appearance | Pseudopolyps; *loss of haustra* ("lead pipe" sign on imaging) | *Cobblestone mucosa*, strictures (thin and string like) |
| Smoking | Protective | Increases risk |

There are many other signs here – see Vinod's lecture and what not for them

**Given the patient's history, you decide to perform a colonoscopy as a screening test and you discover polyps, describe the adenoma-carcinoma sequence and outline the roles of oncogenes, caretaker genes and gatekeeper genes.**

NB. I don't think the adenoma-carcinoma sequence has ever been tested but it's useful to illustrate: 1) multiple mutations must occur before colorectal carcinoma occurs and 2) the role of oncogenes/caretaker genes/gatekeeper genes

1. APC mutation -> increased risk for polyp formations
2. K-ras mutation -> formation of polyp
3. P53 mutation (and increased expression of COX) -> progression to carcinoma

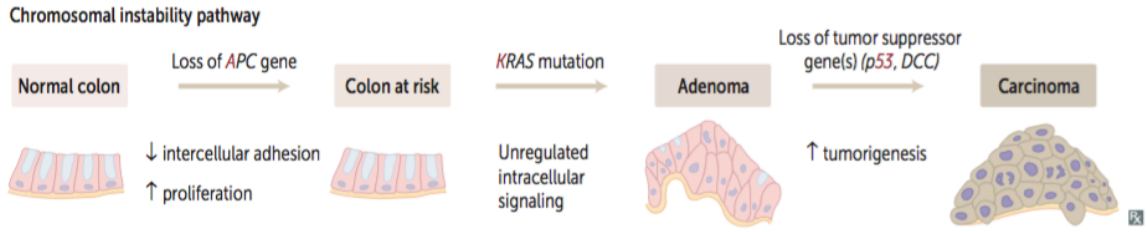
Oncogenes: genes that are important in regulating the cell cycle but, once mutated, will cause cancer (they have the potential to cause cancer but *must* be mutated first)

- APC, K-ras and p53 are all oncogenes

Caretaker genes: genes involved in *DNA repair* and help prevent the accumulation of mutations
Gatekeeper genes: genes encoding proteins that *regulate cell proliferation*

- APC, K-ras and p53 are tumor suppressor genes which prevent a cell from advancing in its cell cycle

(firing order of events is AK-53).



CASE 2: DO YOU EVEN LFT BRO?

NB. things in brackets are low yield but are useful in understanding/remembering how things work

Quick rundown of liver enzymes:

- AST
    - Elevated in **alcohol injury** (aspartate aminotransferase is a mitochondrial enzyme and alcohol is a mitochondrial toxin, hence, alcohol kills mitochondria causing the release of AST into the blood)
- ALT
    - See in **liver damage** (alanine aminotransferase is found in the cytosol of liver cells, hence, necrosis of liver cells cause ALT to be released into the blood)
- ALP
    - Found in **bone and liver disease**
    - Decreased bile flow will result in decreased excretion which will then cause ALP to be released into the blood
    - In periods of bone growth, there will be increased osteoblastic activity which causes elevated ALP (ALP creates an alkaline environment under which osteoblastic activity occurs)
- GGT
    - Think of it as useful in **differentiating bone and liver disease** if ALP is elevated
    - Especially elevated in **bile duct** pathology and in **alcoholics**
    - Can be elevated in a lot of things

Quick rundown of liver markers:

- Bilirubin
    - Some enzyme (UDP-glucuronosyltransferase) within the liver conjugates bilirubin to make it soluble in the blood
    - Depending on where the problem is, there will be high levels of unconjugated/conjugated bilirubin which build up and then overflow into the blood
        - High levels of **unconjugated** bilirubin indicate that there are issues at the level of the liver since it can't conjugate
        - High levels of **conjugated** bilirubin indicate that there is a blockage in the biliary tree since it can't be excreted into the duodenum
- Albumin/platelets
    - Normally produced by the liver (low levels mainly seen in advanced liver disease)

**What changes in liver enzymes would you expect to see in a patient with…**
**Alcoholic hepatitis?**

- AST > ALT, 2:1 – **in most liver disease, the ratio is the opposite – alcoholic liver disease causes AST > ALT**
- Elevated GGT
- Elevated unconjugated bilirubin

**Obstruction of the common bile duct?**

- ALT > AST
- Elevated ALP
- Elevated GGT
- Elevated conjugated bilirubin

**Advanced cirrhosis of liver?**

- AST > ALT
- Normal GGT
- Elevated unconjugated bilirubin

**Viral hepatitis?**

- ALT > AST **(both are in the thousands)**
- Elevated GGT
- Elevated unconjugated bilirubin

**Explain the mechanisms for the following in advanced liver disease:**

| Finding in liver disease | What its caused by | mechanism | Name another disease that causes this finding |
|---|---|---|---|
| Oedema | **Albumin deficiency** | **Less oncotic pressure** | Renal disease: **Nephrotic syndrome** |
| **Elevated prothrombin time** | **Less production of clotting factors** | **Self-explanatory** | GI disease: **pancreatitis, Crohn's, biliary obstruction, coeliac etc.** |
| Confusion, headache | **High levels of ammonia → encephalopathy** | **Direct effect of ammonia on brain** | **----** |

**This should also prompt discussion of other concepts such as portal hypertension, gynaecomastia etc.**