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?

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

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

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

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

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

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

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

Is anything absorbed or chemically digested in the stomach?

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
- H1 antagonist
- Muscarinic agonist

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.

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?

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		
Treated with broad-spectrum antibiotics in hospital for the last 2 weeks **Bonus question: what is the most common bug that causes infection in hospitals following antibiotic use?**		
Greatly elevated serum ALP and GGT, painless jaundice		
Inflammatory changes in small intestinal mucosa, positive anti-endomysium and anti-tissue transglutaminase antibodies		
Recent surgical resection of ileum due to adenocarcinoma		

CASE 3: 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)?

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

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

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		
Location		
Gross appearance		
Effect of Smoking		

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.

CASE 4: 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?

Obstruction of the common bile duct?

Advanced cirrhosis of liver?

Viral hepatitis?

Explain the mechanisms for the following in advanced liver disease:

Finding in Liver Disease	What it is caused by	Mechanism	Name another disease that causes this finding
Oedema			
Elevated prothrombin time			
Confusion, headache			