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1. **Which one of the following is not an effect of cholecystokinin?**
2. It causes gallbladder contraction
3. It increases the rate of gastric emptying
4. It relaxes the sphincter of Oddi
5. It stimulates pancreatic acinar cells
6. It has a trophic effect on pancreatic acinar cells

Case 1

A 53-year-old patient was admitted to the emergency room with acute epigastric pain of 4 hours duration. History reveals that the patient has rheumatoid arthritis for which he takes NSAIDs. The patient also admits to vomiting, which was non-bilious and had a coffee ground consistency. On physical examination there was guarding of the abdominal wall.

1. **What are your differential diagnoses?**
* **Peptic ulcer disease**
1. **What parts of the patient’s history places him at risk of this condition? What are some other risk factors? How do they contribute to the pathogenesis of this condition?**

|  |  |
| --- | --- |
| **Risk factor** | **Rationale** |
| **History of NSAID use** | **Inhibition of both COX 1 and COX 2****Decreases the amount of physiological prostaglandins****Decreases mucus and HCO3- production in the stomach****Less protection of mucosa** |
| **Helicobacter pylori infection** | **Bacteria invades mucosa****Produces urease and inflammatory mediators****Urease converts urea to ammonia which increases the pH to protect the bacteria from stomach acid****But increased pH causes the stomach to produce more acid which damages the mucosa** |
| **Smoking** | **Impaired blood flow to the stomach****Reduces healing of gastric mucosa** |
| **Steroid use** | **Similar effect to NSAIDs****Impairs healing of gastric mucosa** |
| **Zollinger-Ellison syndrome** | **Hypersecretion of acid** |

1. **What is the significance of:**
	1. **Non-bilious vomit?**
* **Vomit doesn’t contain any bile 🡪 bleeding is occurring proximal to the duodenum**
	1. **Coffee ground vomitus?**
* **Blood is partially digested 🡪 distal to the oesophagus**
1. **Fill in the following table with different drugs and their effect on gastric acid secretion:**

|  |  |
| --- | --- |
| **Drug class** | **Effect on gastric acid secretion** |
| **Proton pump inhibitors (e.g. omeprazole)** | * **Irreversibly inhibits the proton pump (H/K ATPase)**
* **Reduces basal and stimulated gastric acid secretion**
* **Almost total inhibition of acid secretion**
* **Proton pump must be resynthesised**
 |
| **Histamine H2 blockers (e.g. ranitidine)** | * **Competitively inhibits histamine H2 receptors**
* **Inhibits basal and food induced acid secretion by 90%**
* **Acid secretion stimulated by histamine and gastrin is blocked**
 |
| **Prostaglandin analogues (misoprostol)** | * **Stable synthetic analogue of prostaglandin**
* **Reduces acid secretion by acting on parietal cells to reduce cAMP levels**
* **Acts on ECL cells to inhibit histamine release**
 |

1. **How do you treat this condition? What are the treatment options if a urea breath test comes back positive?**
* **This patient:**
	+ **Stop taking NSAIDs (if possible)**
	+ **PPI**
* **If urea breath test is positive see table below:**

|  |
| --- |
| **Drugs that treat H. pylori infection** |
| **Triple therapy:**1. ***Antibiotics***

***(amoxycillin, metronidazole or clarithro-mycin)***1. ***PPIs***
2. ***Bismuth chelate***
 | **Antibiotics** | **PPIs** | **Bismuth chelate** |
| * **Need multiple antibiotics to ensure no drug resistant bacteria remain**
* **Monotherapy not effective**
 | * **PPI allows ulcer to heal and increases pH**
	+ **Makes environment less hospitable for bacteria**

 | * **Bismuth chelate is toxic to H. pylori and inhibits its proteolytic enzymes**
* **Prevents adherence of H. pylori to gastric mucosa**
 |

1. **What are some complications if this condition is left untreated?**
* **Perforation (more likely to occur with anterior ulcers)**
	+ **Can see air bubble under diaphragm on erect X-ray (pneumoperitoneum)**
	+ **Pain**
	+ **Could lead to peritonitis!**
* **Bleeding (more likely to occur with posterior ulcers)**
	+ **Iron deficiency anaemia**
	+ **Melena**
	+ **Haematemesis**
1. **What if…**
	1. **This patient went on to have a gastrectomy. What advice would you give them?**
* **Have smaller, more frequent meals; if not, large amounts of undigested food will enter small intestines causing osmotic diarrhoea (dumping syndrome)**
* **Vitamin B12 supplementation**
	+ **Due to lack of intrinsic factor secreted by the stomach**
	+ **Lack of intrinsic factor 🡪 can’t absorb B12**
	+ **Can lead to megaloblastic/macrocytic anaemia**
	1. **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.**
* **Triple whammy is the concurrent use of NSAIDs, ACE inhibitors/ARBs and a diuretic**
	+ **ACEi/ARBs decrease glomerular filtration by causing vasodilation of efferent renal arterioles**
	+ **Diuretics reduce plasma volume which leads to reduced renal blood flow and this may lead to increased serum creatinine concentrations (due to hypovolemia)**
	+ **NSAIDs cause afferent arteriole vasoconstriction → reducing the ability of the kidneys to regulate (increase) glomerular blood flow**
	+ **Therefore, in a triple whammy the kidney is unable to use its normal compensatory mechanisms and may suffer an acute reduction in glomerular filtration that is marked by a rising serum creatinine.**
* **Monitor for acute renal failure!**
	+ **Reduced urine output**
	+ **Tachycardia**
	+ **Hypotension**
	+ **Peripheral oedema**

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.

1. **What is this sign called?**
* **Steatorrhea**
1. **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** |
| **History of chronic epigastric pain radiating to the back, tenderness on abdominal palpation, nausea, vomiting and heavy alcohol intake**  | **chronic pancreatitis 🡪 pancreatic****insufficiency** | **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** **Bonus question: what is the most common bug that causes infection in hospitals following antibiotic use?** | **Bacterial overgrowth****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****(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** |
| **History of diarrhoea and weight loss, positive anti endomysium and anti-tissue transglutaminase antibodies** | **Coeliac disease** | **Villous atrophy 🡪 less absorption 🡪 steatorrhoea** |
| **Recent surgical resection of ileum due to Crohn’s disease**  |  | **No ileum (where bile salts are usually****reabsorbed) 🡪 lack of bile salt****reabsorption 🡪 steatorrhoea** |

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