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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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