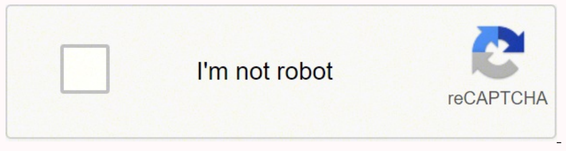

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Case Report

Peptic ulcer disease in pregnancy: A rare cause of rapidly progressing anemia in mid-trimester of pregnancy – A case report and literature review

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ABSTRACT

Peptic ulcer disease is a rare cause of rapidly progressing anaemia in pregnancy, especially during the second trimester of pregnancy. Making a definitive diagnosis in this group of patients is usually very tasking, especially when the common causes of anemia in pregnancy such as Malaria, Sickle Cell Disease, Upper respiratory tract infection, nutritional anemia, e.g., iron and folate deficiency anemia and ruptured ectopic gestation are excluded. We present a rare cause of rapidly progressing mid-trimester severe anemia in pregnancy secondary to peptic ulcer disease in pregnancy, along with the diagnostic challenges, multidisciplinary management, literature review, and the follow-up care.

Key words: Eosophagogastroduodenoscopy; Havana Specialist Hospital Limited; peptic ulcer disease in pregnancy; rapidly progressing severe anemia.

Introduction

Peptic ulcer disease is quite uncommon in pregnancy and a rare cause of rapidly progressing anemia in pregnancy. Making a definitive diagnosis and instituting timely and appropriate management may be quite challenging because of the remarkable anatomical and physiological changes that the gastrointestinal tract (GIT) has undergone during pregnancy and the safety of the investigating tools such as radiological investigations and restriction in drug use during pregnancy.

However, promptness in arriving at a definitive diagnosis and commencing appropriate management will prevent maternal and perinatal morbidities and mortalities in these patients.

This paper reports a rare cause of rapidly progressing severe anemia in mid-trimester of pregnancy, secondary to peptic ulcer disease in pregnancy, in a primigravid woman in a private tertiary health care facility in Lagos, Nigeria.

Case Report

An unbooked 29-year-old G_1Po^{+0} woman at 18 weeks gestation was admitted through our outpatient department with 2-day history of generalized body weakness, vomiting, and poor appetite. There was no history of bleeding per vaginam. She was not a known diabetic and there was no history of peptic ulcer disease. She was a known asthmatic and on inhaler use whenever she had an attack. Her last asthmatic attack was a year before presentation at our facility.

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Peptic Ulcer Disease: Descriptive Epidemiology, Risk Factors, Management and Prevention

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BACKGROUND

Peptic ulcer is a break (like a sore) in the lining of the stomach or the upper part of the small intestine [1], with a diameter of at least 0.5 cm penetrating through the muscularis mucosa. It is typically a non-fatal disease that majorly represented by symptoms of epigastric pain typically relieved by food or alkali, often exhibit periodicity. Peptic ulcers or PUDs are generally categorized based on their anatomical origin as gastric or duodenal. Gastric ulcers are found along the lesser curvature of the stomach, and duodenal ulcers usually occur in the duodenal bulb, the area most exposed to gastric acid [2]. Helicobacter pylori had been thought as the main etiological factor for 90% duodenal and 80% gastric ulcers [3]. With recent decline in prevalence in *H. pylori* in western countries, PUDs, especially gastric ulcers Nonsteroidal Anti-Inflammatory Drugs **(NSAIDs)** and Acetylsalicyclic Acid **(ASA)** [4-5]. In this part of the world, the incidence of duodenal ulcers is approximately four-fold higher than gastric ulcers; constratingly elsewhere, gastric ulcers are more common. Gastric ulcers predominantly arise in subjects over 40 years old whereas,

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Rebleeding after Initial Endoscopic Hemostasis in Peptic Ulcer Disease

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Endoscopic hemostasis is the first-line treatment for upper gastrointestinal bleeding (UGIB). Although several factors are known to be risk factors for rebleeding, little is known about the use of antithrombotics. We tried to verify whether the use of antithrombotics affects rebleeding rate after a successful endoscopic hemostasis for peptic ulcer disease (PUD). UGIB patients who underwent successful endoscopic hemostasis were included. Rebleeding was diagnosed when the previously treated lesion bled again within 30 days of the initial episode. Of 522 UGIB patients with PUD, rebleeding occurred in 93 patients (17.8%). The rate of rebleeding was higher with aspirin medication ($P = 0.006$) and after a long endoscopic hemostasis ($P < 0.001$). Of all significant variables, procedure time longer than 13.5 min was related to the rate of rebleeding (OR, 2.899; 95% CI, 1.768-4.754; $P < 0.001$) on the logistic regression analysis. The rate of rebleeding after endoscopic hemostasis for PUD is higher in the patients after a long endoscopic hemostasis. Endoscopic hemostasis longer than 13.5 min is related to rebleeding after a successful endoscopic hemostasis for PUD.

Keywords: Hemostasis; Endoscopic; Peptic Ulcer; Rebleeding; Upper Gastrointestinal Bleeding

INTRODUCTION

Upper gastrointestinal bleeding (UGIB) remains one of the more common emergencies, despite recent advances in endoscopic techniques. The first-line therapy for UGIB is endoscopic hemostasis. However, even if this treatment is successful, subsequent rebleeding is not a rare event. Studies carried out to determine the predictors of rebleeding in patients with nonvariceal UGIB have revealed the following influential factors such as persistence of endoscopic stigmata, a large ulcer, failure to use a proton pump inhibitor (PPI) after the hemostasis, epinephrine monotherapy, postprocedure use of heparin, and liver cirrhosis (1-3). In addition, a recent Korean study found that the significant risk factors were a lower hemoglobin level (≤ 9 g/dL), a relatively inexperienced therapeutic endoscopist (i.e., a career of < 2 yr), injection of large volumes of epinephrine (> 15 mL), epinephrine monotherapy, and comorbidities such as chronic renal disease or liver cirrhosis (4). However, some lesions rebleed despite the absence of any of these above factors.

While little is known about the effect of antithrombotics on the rate of rebleeding, the worldwide increase in the elderly population means that their use has become an important issue in gastrointestinal (GI) endoscopy. A Japanese study has shown that GI bleeding occurs more frequently in Japanese patients who take aspirin, ticlopidine, and/or warfarin than in those without these antithrombotics (5). That study found that the bleeding occurred in the esophagus or stomach, but not in the lower GI tract. Aspirin is known to irreversibly inhibit the action of cyclooxygenase-1, suppress both tissue prostaglandin synthesis and platelet production of thromboxane A2, and increase the risk of bleeding (6). In addition, warfarin increases the rate of major extracranial hemorrhage, especially when there is a history of GI bleeding, concurrent use of antiplatelet or nonsteroidal anti-inflammatory drugs, genetically different warfarin metabolism, a high international normalized ratio (INR), comorbid illnesses, or a long duration of medication (7). The aim of this study was to determine the risk factors for rebleeding after successful endoscopic hemostasis therapy for peptic ulcer disease (PUD) relative to the use of antithrombotics.

MATERIALS AND METHODS

Patients

UGIB patients due to PUD who underwent successful endoscopic hemostasis between August 2005 and September 2012 at our center were included in this study. Exclusion criteria were the failure of endoscopic hemostasis, cause of bleeding other than PUD, uncertain endoscopic findings, patients under 18 yr-old, or lack of the follow-up data. Bleedings from malignancy were also excluded. The data were collected on the patient's past

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Risk factors leading to peptic ulcer disease: systematic review in literature

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ABSTRACT

This review is aiming to discuss the risk factors which lead to the occurrence of PUD during the period from July 2018 to August 2018. The present review was conducted by searching in Medline, Embase, Web of Science, Science Direct, BMJ journal and Google Scholar for, researches, review articles and reports, published over the past years. Books published on peptic ulcers and on the pathogenesis of human disease were also included., were searched up to August 2018 for published and unpublished studies and without language restrictions, the selected studies were summarized and un reproducible studies were excluded. If several studies had similar findings, we randomly selected one or two to avoid repetitive results. On the basis of findings and results this review found the H. Pylori and the use of NSAIDs are the most common risk factors for developing PUD, and also the genetic, stress and comorbidity increase the risk of PUD occurrence so successful eradication and prevention of the risk factors should be conducted to prevent the presence of PUD and is complication.

Keywords: Peptic ulcer disease, Risk factors, *Helicobacter pylori*, Non-steroidal anti-inflammatory drugs

INTRODUCTION

Peptic ulcer disease (PUD) is a common disease worldwide also known as peptic ulcer or stomach ulcers, PUD occurs as a defect in the mucosa of the stomach or duodenum that exceeds the muscularis mucosa.[1,2] PUD follows gastric mucosal injuries as a result of imbalance between the defensive and the aggressive factors affecting the mucos.[3,4] Many factors contribute to the development of PUD, of which environmental factors such as psychosocial conditions and stress are the most outstanding.[5] Stress is an acute hazard/risk to homeostasis that excites an allostatic or adaptive response. Stress affects the function of the gastrointestinal tract either in short or long-term impacts.[5] Studies revealed that stress contributes to the formation of PUD and is frequently used to produce PUD in experimental animal models.[6] The life time for developing a peptic ulcer is approximately 10%.[7] They resulted in 301,000 deaths in 2013 down from 327,000.[8] In western countries the percentage of people with *Helicobacter pylori* infections roughly matches age (i.e., 20% at age 20, 30% at age

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[PubMed: 20363407]7.Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, Bazzoli F, Gasbarrini A, Atherton J, Graham DY, Hunt R, Moayyedi P, Rokkas T, Rugge M, Selgrad M, Suerbaum S, Sugano K, El-Omar EM., European Helicobacter and Microbiota Study Group and Consensus panel. [PMC free article: PMC6140150] [PubMed: 30228726]2.Lanas Á, Carrera-Lasfuentes P, Arguedas Y, García S, Bujanda L, Calvet X, Ponce J, Perez-Aísa Á, Castro M, Muñoz M, Sostres C, García-Rodríguez LA. Lancet. StatPearls Publishing; Treasure Island (FL): Jul 2, 2021. A gastrin-producing endocrine tumor causes Zollinger-Ellison syndrome or gastrinoma usually arises from the pancreas or duodenum. Investigations Esophagogastroduodenoscopy (EGD): Gold standard and most accurate diagnostic test with sensitivity and specificity up to 90% in diagnosing gastric and duodenal ulcers. The American Society of Gastrointestinal Endoscopy has published guidelines on the role of endoscopy in patients presenting with upper abdominal pain or dyspeptic symptoms suggestive of PUD.[6] Patients over 50 years of age and new onset of dyspeptic symptoms should get evaluated by an EGD. The organism has a wide spectrum of virulence factors allowing it to adhere to and inflame the gastric mucosa. The common causes are persistent H.pylori infection, continued use of NSAIDs, or significant comorbidities that impair ulcer healing or other conditions like gastrinoma or gastric cancer. 2017 Jan 15;11(1):27-37. NSAID-associated PUD Nonsteroidal anti-inflammatory drugs use is the second most common cause of PUD after H. A cluster randomised, crossover, registry-embedded clinical trial of proton pump inhibitors versus histamine-2 receptor blockers for ulcer prophylaxis therapy in the intensive care unit (PEPTIC study): study protocol. Stress reduction counseling can be helpful in some cases.Ulcers are differentiated from erosions based on size. Acute calculous cholecystitis: Review of current best practices. Medications Apart from NSAIDs, corticosteroids, bisphosphonates, potassium chloride, and fluorouracil have been implicated in the etiology of PUD.Smoking also appears to play a role in duodenal ulcers, but the correlation is not linear. Acute ulcers have regular borders, while chronic ulcers have elevated borders with inflammation. An ulcer extends beyond the muscularis mucosa.Signs and symptoms of peptic ulcer disease may vary depending upon the location of the disease and age. Once the protective superficial mucosal layer is damaged, the inner layers are susceptible to acidity. Today, most patients can be managed with a proton pump inhibitor (PPI) based triple-drug therapy.Peptic ulcer disease (PUD) has various causes; however, Helicobacter pylori-associated PUD and NSAID-associated PUD account for the majority of the disease etiology.[1] Causes of Peptic Ulcer Disease Common H. Endoscopy may be required in some patients to confirm the diagnosis, especially in those patients with sinister symptoms. Epigastric pain usually occurs within 15-30 minutes following a meal in patients with a gastric ulcer; on the other hand, the pain with a duodenal ulcer tends to occur 2-3 hours after a meal. First-line treatment for H. pylori infection is more prevalent among those with lower socioeconomic status and is commonly acquired during childhood. [PMC free article: PMC5442405] [PubMed: 28603584]12.Albulushi A, Giannopoulos A, Kafkas N, Dragasis S, Pavlides G, Chatzizisis YS. Fever, tachycardia, positive Murphy sign, leukocytosis, and abnormal liver functions help further distinguish this from biliary colic.[11]These are some potentially life-threatening conditions that can also have similar presentations.Myocardial infarction - especially in the inferior wall and right ventricular involvement, sometimes patients can present with epigastric pain with nausea and vomiting.[12] The presence of other symptoms like dizziness, shortness of breath, and abnormal vital signs in a high-risk patient should alert the clinician to look for this. Mesenteric ischemia - while acute mesenteric ischemia presents with severe, acute onset abdominal pain; the chronic variant usually presents with ongoing post-prandial epigastric pain[13] and can be mistaken for peptic ulcer disease. The pain of gastric ulcers increases 2 to 3 hours after a meal and may result in weight loss, whereas the pain of duodenal ulcers decreases with a meal which can result in weight gain. pylori infectionNSAIDsMedications Rare Zollinger-Ellison syndromeMalignancy (gastric/lung cancer, lymphomas)Stress (Acute illness, burns, head injury)Viral infectionVascular insufficiencyRadiation therapyCrohn diseaseChemotherapy Helicobacter Pylori-Associated PUD H. Only through a team approach can the morbidity of peptic ulcer disease be decreased. Medical Treatment Antisecretory drugs used for peptic ulcer disease (PUD) include H2-receptor antagonists and the proton pump inhibitor (PPIs). Pantoprazole, clarithromycin, and metronidazole, or amoxicillin are used for 7 to 14 days.[7] Antibiotics and PPIs work synergistically to eradicate H. NSAID-induced gastric perforation occurs at a rate of 0.3% per patient per year. NSAIDs block prostaglandin synthesis by inhibiting the COX-1 enzyme, resulting in decreased gastric mucus and bicarbonate production and a decrease in mucosal blood flow. Peptic Ulcer Disease and Helicobacter pylori infection. PPIs have largely replaced H2 receptor blockers due to their superior healing and efficacy. Refractory Disease and Surgical Treatment Surgical treatment is indicated if the patient is unresponsive to medical treatment, noncompliant, or at high risk of complications. A physical exam may reveal epigastric abdominal tenderness and signs of anemia. Corticosteroids, bisphosphonates, and anticoagulants should also be discontinued if possible. The pharmacist should educate the patient on medication compliance to obtain symptom relief and a cure. 2018 Jul;16(7):455-464. The ulcer is round to oval with a smooth base. If it is necessary to use NSAIDs use the lowest possible dose and also consider prophylaxis for patients who use NSAIDs. Obesity has a strong association with peptic ulcer disease, and patients should be asked to lose weight. Curr Gastroenterol Rep. Evaluation and management of acute pancreatitis. It may be used to confirm eradication after 4 to 6 weeks of stopping treatment. Gastric ulcers are commonly located on the lesser curvature between the antrum and fundus. Also, duodenal ulcers are more common in men than in the woman.The peptic ulcer disease (PUD) mechanism results from an imbalance between gastric mucosal protective and destructive factors. This bacterium is responsible for 90% of duodenal ulcers and 70% to 90% of gastric ulcers. [PubMed: 30153780]16.Ayoub F, Khullar V, Banerjee D, Stoner P, Lambrou T, Westerveld DR, Hanayneh W, Kamel AY, Estores D. Mo Med. A refractory peptic ulcer is one over 5 mm in diameter that does not heal despite 8-12 weeks of PPI therapy. It usually occurs in the stomach and proximal duodenum. Older age, presence of risk factors for atherosclerosis, and weight loss should prompt a workup for the same.Mesenteric vasculitis - unexplained abdominal symptoms with or without lower gastrointestinal bleeding in a patient with other features from underlying systemic vasculitis should raise the suspicion of mesenteric vasculitis.[14]The prognosis of peptic ulcer disease (PUD) is excellent after the underlying cause is successfully treated. Prostaglandin analogs (misoprostol) are sometimes used as prophylaxis for NSAID-induced peptic ulcers. [PubMed: 32185509]14.Gnanapandithan K, Sharma A. If first-line therapy fails, quadruple therapy with bismuth and different antibiotics is used. pylorus is a gram-negative bacillus that is found within the gastric epithelial cells. Acute right ventricular myocardial infarction. [PMC free article: PMC7165909] [PubMed: 18837773]5.Lanas A, Chan FKL. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. It extends into the muscularis propria layer of the gastric epithelium. Clin Gastroenterol Hepatol. Gut. The H. 2015 May;13(5):906-12.e2. The role of endoscopy in the management of patients with peptic ulcer disease. This activity reviews the cause, pathophysiology, and presentation of peptic ulcer disease and highlights the role of the interprofessional team in its management. It results in multiple ulcers in the duodenum and jejunum. Treatment may be incorporated with calcium supplements as long-term use of the PPIs can increase the risk of bone fractures. World J Clin Cases. Further, the ability of the mucosal cells to secrete bicarbonate is compromised.H. pylori is known to colonize the gastric mucosa and causes inflammation. [PubMed: 27707777]8.Strand DS, Kim D, Peura DA. 2010 Apr;71(4):663-8. pylori infection.[2][3] The secretion of prostaglandin normally protects the gastric mucosa. 2002 Jan 05;359(9300):14-22. [PMC free article: PMC6511926] [PubMed: 31123673]11.Gomes CA, Junior CS, Di Saverio S, Sartelli M, Kelly MD, Gomes CC, Gomes FC, Corrêa LD, Alves CB, Guimarães SF. [PubMed: 31536217]15.Young PJ, Bagshaw SM, Forbes A, Nichol A, Wright SE, Bellomo R, Bailey MJ, Beasley RW, Eastwood GM, Festa M, Gattas D, van Haren F, Litton E, Mouncey PR, Navarra L, Pilcher D, Mackle DM, McArthur CJ, McGuinness SP, Saxena MK, Webb S, Rowan KM., Australian and New Zealand Intensive Care Society Clinical Trials Group on behalf of the PEPTIC investigators. Alcohol can irritate the gastric mucosa and induce acidity.Hypersecretory environment occurs in the following conditions.Zollinger Ellison syndromeSystemic mastocytosisCystic fibrosisHyperparathyroidismAntral G cell hyperplasiaPeptic ulcer disease (PUD) is a global problem with a lifetime risk of development ranging from 5% to 10%.[4][5] Overall, there is a decrease in the incidence of PUD worldwide due to improved hygienic and sanitary conditions combined with effective treatment and judicious use of NSAIDs.[5] Duodenal ulcers are four times more common than gastric ulcers. pylori also impairs the secretion of bicarbonate, promoting the development of acidity and gastric metaplasia.Gastric ulcers are most commonly located on the lesser curvature, whereas duodenal ulcers are most common at the duodenal bulb. [PubMed: 25460554]3.Huang JQ, Sridhar S, Hunt RH. PPIs block acid production in the stomach, providing relief of symptoms and promote healing. It can be diagnosed by measuring serum gastrin levels.An evidence-based approach to peptic ulcer disease is recommended. Gastroenterology Res. 2018 Jun;11(3):200-206. 2017 Aug 05;390(10094):613-624. Those with gastric outlet obstruction commonly report a history of abdomen bloating and or fullness.Common signs and symptoms include:Epigastric abdominal painBloatingAbdominal fullnessNausea and vomitingWeight loss/weight gainHematemesisMelenaWarning symptoms or alarm symptoms that should prompt urgent referral include:[6]Unintentional weight lossProgressive dysphagiaOvert gastrointestinal bleedingIron deficiency anemiaRecurrent emesisFamily history of upper gastrointestinal malignancyDiagnosis of PUD requires history taking, physical examination, and invasive/non-invasive medical tests. The majority of duodenal ulcers are located in the first part of the duodenum. 6. Surgical options include vagotomy or partial gastrectomy.[9]The following conditions can present with symptoms similar to peptic ulcer disease and it is important to be familiar with their clinical presentation in order to make the correct diagnosis.Gastritis - an inflammatory process of the gastric mucosa from immune-mediated or infectious etiology presenting with upper abdominal pain and nausea. [PMC free article: PMC5997469] [PubMed: 29915630] Any patient presenting with anemia, melena, hematemesis, or weight loss should be further investigated for complications of PUD, predominantly bleeding, perforation, or cancer. Recurrence of the ulcer may be prevented by maintaining good hygiene and avoiding alcohol, smoking, and NSAIDs. Unfortunately, recurrence is common with rates exceeding 60% in most series. pylori-induced PUD is a triple regimen comprising two antibiotics and a proton pump inhibitor. Objectives: Review the causes of peptic ulcer disease.Describe the presentation of a patient with peptic ulcer disease.Summarize the treatment options for peptic ulcer disease.Review the importance of improving care coordination among interprofessional team members to improve outcomes for patients affected by peptic ulcer disease. Surgical treatment of peptic ulcer disease. Gut Liver. 2019 May 06;7(9):1006-1020. Anyone with the presence of alarm symptoms should undergo EGD irrespective of age.Barium swallow: It is indicated when EGD is contraindicated.Complete blood work, liver function, and levels of amylase and lipase.Serum gastric is ordered if Zollinger Ellison syndrome is suspected.Helicobacter pylori testing: Serologic testing Urea breath test: High sensitivity and specificity. 25 Years of Proton Pump Inhibitors: A Comprehensive Review. [PubMed: 11809181]4.Snowden FM. Management of Helicobacter pylori infection-the Maastricht V/Florence Consensus Report. Expert Rev Cardiovasc Ther. Today, testing for Helicobacter pylori is recommended in all patients with peptic ulcer disease. [PubMed: 28242110]6.ASGE Standards of Practice Committee. pylori.[8] The antibiotic selected should take into consideration the presence of antibiotic resistance in the environment. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Nocturnal pain is common with duodenal ulcers. 2017 Jan;66(1):6-30. 2008 Oct;225:9-26. 2017 May 27;9(5):118-126. Mesenteric Vasculitis. StatPearls [Internet]. Risk factors predisposing to the development of PUD:H. In the presence of urease, an enzyme produced by H.pylori, the radiolabeled carbon dioxide produced by the stomach is exhaled by the lungs.Antibodies to H.pylori can also be measured. H. When left untreated, it has significant morbidity. [PubMed: 2072800]10.Chatila AT, Bilal M, Guturu P. 2018 Sep;20(3):182-189. Med Clin North Am. 1991 Jul;75(4):999-1012. Once Versus Twice-Daily Oral Proton Pump Inhibitor Therapy for Prevention of Peptic Ulcer Rebleeding: A Propensity Score-Matched Analysis. Emerging and reemerging diseases: a historical perspective. Review Article: Mesenteric Ischemia. 2018 May-Jun;115(3):219-224. Because the presentation of PUD is often vague, healthcare workers, including nurses, need to be aware of this diagnosis. Biopsies from at least 4-6 sites are necessary to increase sensitivity. Peptic ulcer disease is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. 2020 Mar 17;22(4):17. Crit Care Resusc. PUD is a very common disorder that affects millions of people. The abdominal pain can mimic a number of other pathologies and consequently lead to a delay in treatment.Once the diagnosis is made, the key is to educate the patient on lifestyle changes, which include discontinuation of smoking, abstaining from alcohol and caffeinated beverages, and avoid consumption of too many NSAIDs. Gastroenterology nurses monitor patients, provide education, and keep the team updated on the patient's condition. Therefore, in patients with a history of PUD, COX-2 selective NSAIDs are preferred. World J Gastrointest Surg. Immunol Rev. Access free multiple choice questions on this topic. Computerized tomography of the abdomen with contrast is of limited value in the diagnosis of PUD itself but is helpful in the diagnosis of its complications like perforation and gastric outlet obstruction. This results in hypochlorhydria or achlorhydria, leading to gastric ulceration. Virulence Factors of Helicobacter PyloriUrease: The secretion of urease breaks down urea into ammonia and protects the organism by neutralizing the acidic gastric environment.Toxins: CagA/VacA is associated with stomach mucosal inflammation and host tissue damage.Flagella: Provides motility and allows movement toward the gastric epithelium. It may involve the lower esophagus, distal duodenum, or jejunum. A careful history should be obtained and noted for the presence of any complications. Stool antigen test Urine-based ELISA and rapid urine test Endoscopic biopsy: Culture is not generally recommended as it is expensive, time-consuming, and invasive. [PMC free article: PMC5221858] [PubMed: 27840364]9.Sachdeva AK, Zaren HA, Sigel B. NSAIDs induced PUD can be treated by stopping the use of NSAIDs or switching to a lower dose. Lesions less than 5 mm in diameter are termed erosions, whereas lesions greater than 5 mm in diameter are termed ulcers. Patient reporting of epigastric abdominal pain, early satiety, and fullness following a meal raise suspicion of PUD. Peptic ulcer disease (PUD) is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. If the ulcer persists despite addressing the above risk factors, patients can be candidates for surgical treatment. Banerjee S, Cash BD, Dominitz JA, Baron TH, Anderson MA, Ben-Menachem T, Fisher L, Fukami N, Harrison ME, Ikenberry SO, Khan K, Krinsky ML, Maple J, Fanelli RD, Strohmeyer L. A dietary consult should be sought as there is evidence that obesity may be a trigger factor for peptic ulcer disease. Gastric and duodenal ulcers can be differentiated from the timing of their symptoms in relation to meals. The majority of patients with PUD present to their primary caregiver, but others may present to the emergency department, urgent care clinic, or an outpatient clinic. COX-2 selective NSAIDs are less likely to cause PUD as COX-2 is not expressed on the gastric mucosa. It is indicated if eradication treatment fails or there is suspicion about antibiotic resistance. Gastrointest Endosc. pylori infectionNSAID useFirst-degree relative with PUDEmigrant from a developed nationAfrican American/Hispanic ethnicityWith peptic ulcers, there is usually a defect in the mucosa that extends to the muscularis mucosa. For most patients with PUD who are treated with the triple regimen or PPI, the outcomes are excellent, but recurrence of symptoms is not uncommon.[15][16] (Level 2)Review Questions1.Narayanan M, Reddy KM, Marsicano E. Peptic ulcer disease. [PubMed: 29902098]13.Gnanapandithan K, Feuerstadt P. Clinical presentation is very similar to that of peptic ulcer disease.Gastroesophageal reflux disease (GERD) - patients usually describe a burning sensation in the epigastrium and lower retrosternal area, excessive salivation, or intermittent regurgitation of food material.Gastric cancer - apart from abdominal pain, patients usually describe alarm symptoms like weight loss, melena, recurrent vomiting, or evidence of malignancy elsewhere in case of metastasis.Pancreatitis - epigastric or right upper quadrant pain that is more persistent and severe, worse in the supine position, and patients usually have a history of alcoholism or gallstones.[10] Elevated serum amylase and lipase are useful in the diagnosis.Biliary colic - intermittent, severe deep pain in the right upper quadrant or epigastrium precipitated by fatty meals.Cholecystitis - right upper quadrant or epigastric pain that usually lasts for hours and is exacerbated by fatty meals and is associated with nausea and vomiting. Following complications can occur in PUD:Upper gastrointestinal bleedingGastric outlet obstructionPerforationPenetrationGastric cancerPatients with peptic ulcer disease (PUD) should be counseled about potentially injurious agents like nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, alcohol, tobacco, and caffeine. However, unlike in the past, mortality rates for peptic ulcer disease have decreased significantly.Peptic ulcer disease (PUD) if not diagnosed and treated promptly can lead to serious complications.

01/07/2021 · Recognize the symptoms of an ulcer. Symptoms of stomach ulcers vary from person to person. If you believe you have an ulcer but haven’t had it diagnosed by a medical professional, see your doctor. The symptoms of a stomach ulcer include: Burning pain in the area just below the ribcage in the center of your chest. 01/04/2020 · Gastroesophageal reflux disease (GERD) is mainly a clinical diagnosis based on typical symptoms of heartburn and acid regurgitation. Current guidelines indicate that patients with typical symptoms should first try a proton pump inhibitor (PPI). If reflux symptoms persist after 8 weeks on a PPI, endoscopy of the esophagus is recommended, with biopsies taken to ... Excessive sodium intake is a major public health concern globally. 1, 2 Numerous studies have found high sodium intake to be associated with increased risks of cardiovascular disease (CVD) and all-cause mortality among patients with hypertension, 3–7 but findings among individuals without hypertension have been equivocal. 3–12 Although several randomized controlled trials ...

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