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In this section: What is a peptic ulcers? A peptic ulcers? Researchers estimate about 1% to 6% of people in the United States have peptic ulcers.1 Who is more likely to develop peptic ulcers? People are more likely to develop peptic ulcers if they are the two most common causes of peptic ulcers. People are more likely to develop peptic ulcers if they are older adults had a peptic ulcer before smoke What are the complications of peptic ulcers? Peptic ulcers can lead to complications such as bleeding in your stomach or duodenum, which can lead to peritonitis, an infection of the lining of the abdominal cavity penetration of the ulcers through the stomach or duodenum and into another nearby organ a blockage that can stop food from moving from your stomach into your duodenum Reference [1] Ingram RJM, Ragunath K, Atherton JC. Chapter 56: Peptic ulcer disease. In: Podolsky DK, Camilleri M, Fitz G, et al, eds. Yamada's Textbook of Gastroenterology. 6th ed. John Wiley & Sons, Ltd; 2016:1032-1077. This content is provided as a service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), part of the National Institutes of Health. NIDDK translates and disseminates research findings to increase knowledge and understanding about health and disease among patients, health professionals, and the public. Content produced by NIDDK is carefully reviewed by NIDDK scientists and other experts. Peptic ulcers may cause symptoms of indigestion. Common symptoms of indigestion and breastbone feeling full too soon while eating a meal feeling uncomfortably full after eating a meal nausea and vomiting bloating belching Abdominal pain is the most common symptom of a peptic ulcer. Abdominal pain is the most common symptom of a peptic ulcer. The pain may be dull or burning and may come and go over time. For some people, the pain may be dull or burning and may go away for a short time after they eat. For other people, eating may make the pain worse. Many people who have symptoms includers don't have any symptoms. They may not develop symptoms until an ulcer leads to complication. These symptoms include a symptom includers don't have any symptoms that could be caused by a complication. These symptoms include a symptom includers don't have any symptom include black or tarry stool, or red or maroon blood mixed with your stool red blood in your vomit or vomit that looks like coffee grounds sudden, sharp, or severe abdominal pain that doesn't go away feeling dizzy or fainting a rapid pulse or other symptoms of shock a change in or worsening of your peptic ulcer symptoms What causes peptic ulcers? The most common causes of peptic ulcers are Helicobacter pylori (H. pylori) infection and nonsteroidal anti-inflammatory drugs (NSAIDs). Other causes of peptic ulcers are uncommon or rare. People with certain risk factors are more likely to develop ulcers. H. pylori H. pylori H. pylori infection is a common cause of peptic ulcers. Researchers are still studying how people become infected with H. pylori bacteria may spread from person to per naproxen—is another common cause of peptic ulcers. NSAIDs relieve pain, but they also make the stomach lining more prone to damage and ulcers. You have a higher chance of developing a peptic ulcer due to NSAIDs if you take NSAIDs for a long time a type of NSAID that is more likely to cause an ulcer high doses of an NSAID or more than one NSAID NSAIDs along with other medicines that increase the risk for ulcers include infections caused by certain viruses, fungi, or bacteria other than H. pylori medicines that increase the risk of developing ulcers, including corticosteroids, medicines used to treat low bone mass, and some antidepressants, especially when you take these medicines with NSAIDs surgery or medical procedures that affect the stomach or duodenum Less common causes of peptic ulcers also include certain diseases and health conditions, such as In rare cases, doctors can't find the cause of peptic ulcers. Doctors may call ulcers with unknown causes idiopathic peptic ulcers. As a library, NLM provides access to scientific literature. Inclusion in an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice Peptic ulcer is a lesion of the mucosal lining of the mucosal lining of the mucosa, having H. pylori as the main etiologic factor. Dietotherapy is important in the prevention and treatment of this disease. To update nutritional therapy in adults' peptic ulcer. Exploratory review without restrictions with primary sources indexed in Scielo, PubMed, Medline, ISI, and Scopus databases. Dietotherapy, as well as caloric distribution, should be adjusted to the patient's needs aiming to normalize the nutritional status and promote healing. Recommended nutrients can be different in the acute phase and in the recovery phase, and there is a greater need of protein and some micronutrients, such as vitamin C in the recovery phase. In addition, some studies have shown that vitamin C in the recovery phase. In addition, some studies have shown that vitamin C in the recovery phase. In addition, some studies have shown that vitamin C in the recovery phase. In addition, some studies have shown that vitamin C in the recovery phase. because they reduce the side effects of antibiotics and help reduce treatment time. A balanced diet is vital in the treatment of peptic ulcer, once food can prevent, treat or even alleviate the symptoms involving this pathology. However, there are few papers that innovate dietotherapy; so additional studies addressing more specifically the dietotherapy for treatment of peptic ulcer are necessary. Keywords: Nutrition; Dietotherapy; Helicobacter pylori; Nutritional therapy; Helicobacter pylori; Nutritional therapy; treatment A úlcera péptica é uma lesão que ocorre na mucosa do trato gastrointestinal, sendo caracterizada por um desequilíbrio entre fatores agressores e protetores da mucosa gástrica, tendo como principal fator etiológico o H. pylori. A dietoterapia é fundamental na prevenção e tratamento dessa patologia. Rever a terapia nutricional na úlcera péptica em adultos. A metodologia utilizada foi um estudo exploratório de revisão do conhecimento disponível na literatura científica. A dietoterapia bem como a distribuição calórica deve ser ajustada as necessidades do paciente com objetivo de normalizar o estado nutricional e promover a cicatrização. As recomendações de nutrientes podem ser diferenciadas nas fases aguda e de recuperação, havendo uma maior necessidade proteica e de alguns micronutrientes como vitamina A, zinco, selênio e vitamina C na fase de recuperação. Além disso, alguns estudos evidenciam que a vitamina C tem efeito benéfico na erradicação do H. pylori. As fibras e probióticos também possuem um importante papel no tratamento da úlcera péptica, reduzindo os efeitos colaterais dos antibióticos e auxiliando na redução do tempo de tratamento da úlcera péptica, reduzindo os efeitos colaterais dos antibióticos e auxiliando na redução do tempo de tratamento. Percebe-se que poucos são os trabalhos que evidenciam a terapia nutricional da úlcera e não há consenso sobre o tema. Com isso, mais estudos são necessários para abordar com maior especificidade o tratamento dietoterápico da úlcera péptica. Dieta equilibrada é fundamental no tratamento da úlcera péptica, uma vez que o alimento pode prevenir, tratar ou mesmo aliviar os sintomas que envolvem esta doença. No entanto, existem poucos trabalhos que inovam dietoterapia; assim, são necessários estudos adicionais abordando mais especificamente a dietoterapia para o tratamento de úlcera péptica. Peptic ulcer is a disease of chronic development, characterized by an imbalance between the factors that damages the mucosa and those for its protection, resulting in a lesion of the lining of the upper digestive tract22. It has been one of the most prevalent diseases in the world, and some of its complications have been the major causes of morbidity and mortality34. The prevalence differs in the world population between the duodenal and gastric ulcers, and the mean age of people with the disease is between 30 and 60 years, but it can happen in any age. Racial difference has also been observed, and in Africa duodenal ulcers are found to be rare in black people, but in the United States the incidence is the same for blacks and whites; regarding gender, there is predominance of ulcers in males 16. Peptic ulcer has a multifactor etiology. Environmental elements such as alcohol and nicotine can inhibit or reduce secretion of mucus and bicarbonate, increasing acid secretion. Genetic factors can influence, and children of parents with duodenal ulcer are three times more likely to have ulcer than the population 14. In the past decades, the identification of Helicobacter pylori and ulcers associated with the chronic use of antiinflammatory drugs contributed to a better understanding of the events associated to the genesis of peptic ulcers36. Nutrition and its recommendations define aspects of a healthy diet, and the need to establish nutritional benchmarks is long recognized as a way to promote health and prevent and treat diseases. Accordingly, dietotherapy has played as key role in the prevention and treatment of Peptic ulcer, with the main purpose of recovering and protecting the gastrointestinal lining, improving digestion, relieving pain, and contributing to a satisfactory nutritional status 29. Peptic ulcer is a disease known since antiquity, but there are few studies innovating dietotherapy as treatment for this disease. For this reason, the objective of this study was to review nutritional therapy of peptic ulcers in adults. Review of the knowledge available in the scientific literature about nutritional therapy of peptic ulcer, without restrictions of date, based on Scielo, PubMed, Medline, ISI, and Scopus databases. Was also included data from national and international health committees. For the search in databases, were used the following descriptors: dietotherapy, nutrition, peptic ulcer, Helicobacter pylori, peptic ulcer, Helicobacter pylor mucosa exposed to chloride peptic secretion. It often occurs in the duodenum (5-10% of the population), stomach or esophagus36. It is a chronic disease, with activation and remission periods and its pathogenesis is characterized by the imbalance between the factors that damages the mucosa (chloride acid, pepsin, and ulcerogenic drugs) and those that protect it (mucosal barrier, prostaglandins, and mucosal secretion)18. Clinical manifestations are characterized by epigastric discomfort, burning or severe and continuous pain, which tends to be worse at night. Pain usually happens one to three hours after eating, and may be followed by nauseas, vomiting, discomfort in the gastrointestinal tract, flatulence, and significant loss of body weight22. Important factors in the etiopathogenesis are tobacco, alcohol, and Helicobacter pylori, which is able to move in high viscosity medium, adhering to the mucosa epithelium, where it remains protected18. Diagnosis of this infection may be achieved through various tests, each with a sensitivity and specificity above 80%. The golden standard test is the upper endoscopy, which allows the physician to collect material to check for the presence of H. pylori besides other therapeutic procedures 36. It aims to identify possible nutritional alterations and determine proper intervention to ensure the individuals' health. Malnutrition in this case may occur especially when there is stenosis, which prevents normal ingestion of foods18. For nutritional assessment, some important indicators are used in this process, such as the anthropometric, biochemical, and clinical evaluations. The anthropometric assessment consists of weight and height measurements that may be used in conjunction in the assessment of the nutritional status by means of BMI (Body Mass Index), but this method does not distinguishes losses of fat or lean mass. In addition, weight may be concealed by hyper-hydration or de-hydration, thus not resulting in an accurate determination of the nutritional status in these specific cases 18. Total body bioelectrical impedance is a method used to measure the body mass, liquid volume, and body fat, being recognized by the Brazilian Ministry of Health and the Food and Drug Administration as a valuable technique for this purpose 14. Indirect calorimetry is a non-invasive method to determine the nutritional needs and the utilization rate of energy substrates from oxygen consumption and carbon dioxide production obtained by analysis of the inhaled and exhaled air by the lungs8. The upper arm muscle circumference is a measure to assess somatic protein compartment, and the corrected muscle air by the lungs8. The upper arm muscle circumference is a measure to assess somatic protein compartment, and the corrected muscle air by the lungs8. appropriately. The triceps skinfold is the most used skinfold because it is the triceps region that best represents the adipose subcutaneous layer28. Biochemical tests are able to diagnose possible deficiencies still in the subclinical phase and includes serum albumin, which plays a key role in the nutritional assessment - serum pre-albumin a sensitive indicator of protein deficiency -, having several advantages to help determine the nutritional status and intervention needs18. Complete blood test is often used in this case, because it involves counts of white and red blood cells, reticulocytes and platelet, hematological indices, thus allows to monitor blood alterations and a progress analysis of the disease 18. Nitrogen balance is a noninvasive and accessible technique consisting of the difference between taken-in oxygen and excreted oxygen used to assess protein intake and protein degradation 17. The objective of peptic ulcer dietotherapy is to prevent hyper secretion of peptic chloride in order to reduce the sore and pain in the gastric and duodenal mucosa. In addition, nutritional therapy aims to promote healing, based on a complex sequence of events going from the initial trauma to the repair of the damaged tissue. Investigation of nutritional deficiencies is essential in the preparation of an appropriate recovery diet. In the early 20th century, Sippy proposed a diet based on milk and milk cream, combined with antacids, for treatment of gastrointestinal ulcer, based on the principle that milk would provide gastric acid secretion effect of milk29. According to Marrota and Floch18, calories distribution for patients with peptic ulcer should be normal, with values ranging from 50-60% of carbohydrates, 10-15% of proteins, and 25-30% of lipids, with total energy value sufficient to maintain or recover the nutritional status. Reis29 suggested that calories distribution should be adjusted according to the patient's needs to normalize the nutritional status, having as recommended macronutrients a protein intake of up to 1.2 g/kg/weight/day in the acute stage (5th to 8th week) and up to 1.5 g/kg/weight/day in the recovery stage. Carbohydrates should be adjusted to the patient's needs, without disaccharides concentration, so as to avoid fermentation, and lipids without concentration of saturated fats. To accelerate the healing process, in addition to protein, there are specific micronutrients such as zinc, which is essential to maintain the immune system function, as a response to oxidative stress, and to heal wounds25. Selenium may reduce infection complications and improve healing10. In addition, vitamin A may be used as a supplement, but the research that supports this practice is of limited effectiveness, because very high dosages do not promote cure, and excessive intake may be toxic2. Nutritional recommendations for patients with peptic ulcer are described in Table 1. Recommended daily diet for peptic ulcer Characteristics Recommendations Daily energy needs (DEN) Sufficient to maintain or recover the nutritional status 20-25 Kcal/Kg: weight loss 25-30 Kcal/Kg: weight loss 25-30 Kcal/Kg: weight loss 25-30 Zinc (mg)13 11 40 Selenium (μg)20 55 400 Vitamin A (μ g)113 900 3000 Vitamin C (μ g)13 40 400 Iron (μ g)13 2.4 Folic acid (μ g)13 2.4 Folic acid (μ g)13 40 400 Iron (μ g)13 45 45 Fibers (μ g)13 20 to 30 Probiotics (UFC/day)18 109 to 1011 lactic acid bacteria 109to can seen that the authors agree with the recommendations to improve healing, differing only when the patient is in the active or remission phase. Therefore, to help plan a more specific and safe action, it is important to investigate the individual's nutritional status and if the patient has any associated pathology. The physicochemical properties of fiber fractions produce different physiological effects in the organism. Soluble fibers, found in apple, oatmeal, and pear are responsible, for instance, for an increased viscosity in the intestinal content. Insoluble fibers (whole grains, granola, flaxseed) increase stool bulk, reduce transit time in the large intestine, and make fecal elimination easier and quicker. Fibers regulate the bowel function, which make them vital for the well being of healthy people and in the dietary treatment of many pathologies 19. Räihä et al.26 reported a large number of patients with peptic ulcer is advisable (20 to 30 g/day, according to WHO - World Health Organization), because fibers act as buffers, reducing concentrations of bile acids in the stomach and the intestinal tract18. Probiotics are defined as a food supplement based on live microorganisms, which affect beneficially the human organism by providing a microbial balance33. There is a special interest in probiotics for treating infection by H. pylori, because it plays a crucial role in the pathogenesis of chronic gastritis and peptic ulcer in adults9. Probiotics have therapeutic agents against H. pylori, which can be shown by clinical data that prove the efficacy of some probiotics in diverse gastrointestinal diseases and also due to the increasing resistance of pathogenic bacteria to antibiotics35. One of the measures that may contribute to reduce the infection rate by H. pylori, but have the ability to reduce the bacterial load and infection in animals and humans 32. Studies on humans indicate that probiotics improve slightly the elimination rate in treatment against H. pylori, being useful to decrease the bacterial load and likely improve dyspeptic symptoms 37. Thus, an intake of 10° to 10¹¹ CFU/day of lactic acid bacteria is recommended. Among the clinical applications of probiotics, reduction of the side effects associated with H. pylori receiving L. acidophilus (108 CFU) for three weeks, it was capable of inhibiting the growth H. pylori in 64% of the volunteers. Similarly, in a study by Wang et al.38 with 59 volunteers, they received Bifidobacterium animalis and L. acidophilus (1010 CFU) twice a day during six weeks, and concluded that regular intake of yogurt containing Bifidobacterium animalis and L. acidophilus can effectively suppress the infection caused by H. pylori in humans. Some authors show that the best treatment is the eradication of the bacteria 8. Accordingly, some studies in humans40used antioxidants to eradicate H. Pylori and observed that vitamin C for a longer period of time had a better response when compared with higher doses. Thus, it is observed that patients with peptic ulcer by H. pylori can take up to 500 mg, according to DRIs13. Another antioxidant used to eradicate H. pylori is the capsaicin present in pepper and chilies. Studies on animals showed that capsaicin has effect in healing gastrointestinal lesions. Likewise, some researchers39 studied the effect of capsaicinoids in individuals with aspirin-induced lesions. It is worth noting that peppers may be associated with irritations in the gastric mucosa, and may not have a gastroprotective effect in some individuals with peptic ulcer. According to César et al.5, damages caused by ulcer can be reverted, often after treatment of an infection caused H. Pylori, by changing diet and lifestyle. Ferri-De-Barros et al.11 observed that alcohol consumption causes damages to the digestive tract with appearance of symptoms of ulcer and others. According to Reis29, smoking diminishes secretion of mucus and bicarbonate, raising the duodenal and gastric flow and increasing the risk of ulcers formation. Prospective and retrospective studies show higher mortality from peptic ulcer in smokers when compared to nonsmokers. Researches show that, among other tobacco constituents, nicotine accounts for most of the peptic ulcer development, because it has a harmful effect on the protective mucus of the gastric epithelium, altering bicarbonate 30. Coffee, even decaffeinated coffee, raises gastric acid production, resulting in mucosal irritations. The same goes for soft drinks, which, besides increasing acid production, are gaseous and cause gastric distension and is dyspepsia-related 18. However, it is important to take into account individual tolerances, with attention to the fermented milk Fatty cheeses (mascarpone, cream cheese, gorgonzola) - Oilseeds Flaxseed, Brazilian nut, walnuts - Oils and olive oils Vegetables Leafy dark green vegetables, carrot, beet, green bean, spinach, kale, radish zucchini, leek Broccoli, cauliflower, cabbage, cucumber, onion, red pepper Spicy peppers (black pepper Spicy peppers (black pepper, chilies) Legumes Bean soup, lentils, chickpeas, soybean Beans - Meats Lean meat (beef, pork, chicken, fish) Fatty meats, organ meats and sausages - Sweets - Concentrated sweets Chocolate Beverages Natural juices Citrus/acidic fruit juices Coffee, black tea, fizzy/cola drinks Other foods - Industrialized seasonings, spices and condiments (Ketchup, mayonnaise, mustard) Mustard grain Deficiency of vitamin B12 is common in patients with peptic ulcer due to the prolonged use of antacids, making difficult the bioavailability of this vitamin. Vitamin B12 can be synthetized by the intestinal microbiota in the colon, but is not absorbed. Deficiency of this vitamin and megaloblastic anemia. It is estimated that 80-90% of patients lacking vitamin, which can be obtained from animal foods, such as milk, meat and eggs. Absorption of folic acid can be impaired in subjects that make chronic use of aluminum-based antacids (Pepsamar®, Gastran®, Alca-Luftal®), because antacids make the pH of the jejunum more alkaline23. In these cases, intake of 400 µg/day of this vitamin is necessary, which can be supplied with ingestion of leguminous foods such as lentils, and meats. It is important to emphasize that the reduction of the gastric acidity by antacids or antiulcers (Lanzol®, Prazol®, Omeoprazol®) alters proteins digestion and affects good digestion of foods21. Antacids can also diminish absorption of iron, causing iron-deficiency anemia. Gastrointestinal bleeding can be observed in gastroduodenal ulcer and infection by H. pylori1 and may be associated with the development of anemia. Gastric bleeding is a major complication of peptic ulcer22. Infection by H. pylori can also lead to an imbalance of body iron homeostasis due to the growing demand for iron by the same. Similarly to other kinds of bacteria, iron is essential for the growth of H. pylori1. To prevent or even treat iron deficiency, an intake of 45 mg of iron daily is recommended, which can be supplied by the ingestion of meats, the main source of heme iron. It is estimated that 100 g of meat correspond to 1 kg of beans (non-heme iron). The concomitant consumption of fruit juice containing vitamin C enhances the non-heme iron absorption from the diet1. The potential of plants as source of new drugs still offers a large field for scientific research. Even if is observed a large number of known plants, a small percentage has already been phytochemically investigated and only a fraction of them has already been assessed to determine its pharmacological potential Even among traditional medicinal plants there is still a large percentage that has not been studied to confirm their efficacy and safety in humans27. In peptic ulcer this is also observed. In a study conducted by Mentz and Schenkel20, in which they assessed plants with popularly known effects to scientifically prove them, they observed that plants like Symphytum Officinale L. (Comfrey), besides having no proven efficacy it may be harmful because of their pyrrolizidine alkaloids, of proven hepatotoxic action. Another studied plant was Zantoxylon rhoifoliun Lan ("mamica-de-cadela"), popularly indicated for ulcers and healing, but its benefits have not been proved either. In addition, Maytenus ilicifolia Mart, commonly known in Brazil as "espinheira-santa", used for healing peptic ulcer, has not proven this effect in trials either. Studied the effects of Peumus boldus ("boldo") and Baccharis genistelloides ("carqueja"), both commonly used to treat digestive problems and ulcers. The studies that proved various activities popularly attributed to these plants are associated with the isolate chemical compounds, such as, for example, flavonoids, antioxidants found both in boldo and carqueja leaves. However, the benefits of these teas in the cure of peptic ulcer have not been scientifically proven27. The use of natural products in treatment of ulcer has been widely studied. However, most of the studies7that have proven an anti-ulcer effect were conducted with animals, and therefore do not provide reliability for alternative treatment of peptic ulcer, once food can prevent, treat or even alleviate the symptoms involving this pathology. 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The efficacy of Helicobacter pylori eradication regimen with and without vitamin C supplementation. Digestive and Liver Disease. 2009;41:644-647. doi: 10.1016/j.dld.2008.09.008. [DOI] [PubMed] [Google Scholar] Articles from Arquivos Brasileiro de Cirurgia Digestiva : ABCD = Brazilian Archives of Digestiva : ABCD = Brazilian Archives : ABCD = Braz inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. It extends into the muscularis propria layer of the gastric epithelium. It usually occurs in the stomach and proximal duodenum, or jejunum. 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. Today, testing for Helicobacter pylori is recommended in all patients with a duodenal ulcer disease. Endoscopy may be required in some patients to confirm the diagnosis, especially in those patients with sinister symptoms. Today, most patients can be managed with a proton pump inhibitor (PPI) based triple-drug therapy. URL of this page: Also called: Duodenau ulcer, Gastric ulcer, Stomach ulcer, Ulcer A peptic ulcer, Stomach pain is the most common symptom. The pain: Starts between meals or during the night Briefly stops if you eat or take antacids Lasts for minutes to hours Comes and goes for several days or weeks Peptic ulcers happen when the acids that help you digest food damage the walls of the stomach or duodenum. The most common cause is infection with a bacterium called Helicobacter pylori. Another cause is the long-term use of nonsteroidal anti-inflammatory medicines (NSAIDs) such as aspirin and ibuprofen. Stress and spicy foods do not cause ulcers, but can make them worse. To see if you have an H. pylori infection, your doctor will test your blood, breath, or stool. Your doctor will test your stomach and duodenum by doing an endoscopy or x-ray. Peptic ulcers will get worse if not treated. Treatment may include medicines to reduce stomach acids or antibiotics to kill H. pylori. Antacids and milk can't heal peptic ulcers. Not smoking and avoiding alcohol can help. You may need surgery if your ulcers don't heal. NIH: National Institute of Diabetes and Digestive and Kidney Diseases Stomach ulcer (Medical Encyclopedia) Also in Spanish Ulcers (Nemours Foundation) Also in Spanish The information on this site should not be used as a substitute for professional medical care or advice. Contact a health care provider if you have questions about your health. Learn how to cite this page Peptic ulcer disease is characterized by discontinuation in the inner lining of the gastric epithelium. It usually occurs in the stomach and proximal duodenum. It may involve the lower esophagus, distal duodenum, or jejunum. This activity reviews the cause, pathophysiology, and presentation of peptic ulcer disease and highlights the role of the interprofessional team in its management. Objectives: Review the causes of peptic ulcer disease. Review the importance of improving care coordination among interprofessional team members to improve outcomes for patients affected by peptic ulcer disease. Access free multiple choice questions on this topic. Peptic ulcer disease (PUD) is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. It extends into the muscularis propria layer of the gastric epithelium. It usually occurs in the stomach and proximal duodenum, or jejunum. Epigastric pain usually occurs within 15-30 minutes following a meal in patients with a duodenum, or jejunum. Epigastric pain usually occurs within 15-30 minutes following a meal in patients with a duodenum, or jejunum. after a meal. Today, testing for Helicobacter pylori is recommended in all patients with peptic ulcer disease. Endoscopy may be required in some patients to confirm the diagnosis, especially in those patients with sinister symptoms. Today, most patients to confirm the diagnosis, especially in those patients to confirm the diagnosis, especially in those patients with sinister symptoms. (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. 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. pylorus is a gram-negative bacillus that is found within the gastric ulcers. H. pylori infection is more prevalent among those with lower socioeconomic status and is commonly acquired during childhood. The organism has a wide spectrum of virulence factors, allowing it to adhere to and inflame the gastric mucosa. This results in hypochlorhydria or achlorhydria or achlorhydr 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. NSAID-associated PUD Nonsteroidal anti-inflammatory drug use is the second most common cause of PUD after H. pylori infection.[2][3] The secretion of prostaglandin normally protects the gastric mucosa. NSAIDs block prostaglandin synthesis by inhibiting the COX-1 enzyme, resulting in decreased gastric mucosa. NSAIDs block prostaglandin synthesis by inhibiting the COX-1 enzyme, resulting in decreased gastric mucosa. 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. 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 in men than in women. The peptic ulcers are more common in men than in women. The peptic ulcers are more common in men than in women. The peptic ulcers are more common in men than in women. The peptic ulcers are more common in men than in women. The peptic ulcers are more common in men than in women. The peptic ulcers are more common than gastric mucosal protective and destructive factors. Risk factors predisposing to the development of the peptic ulcers are more common than gastric mucosal protective and destructive factors. PUD:H. 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. Once the protective superficial mucosal layer is damaged, the inner layers are susceptible to acidity. Further, the ability of the mucosal cells to secrete bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. H. pylori also impair the secretion of bicarbonate is compromised. most common at the duodenal bulb. The ulcer is round to oval with a smooth base. 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 on the location of the disease and age. Gastric and duodenal ulcers can be differentiated from the timing of their symptoms in relation to meals. Nocturnal pain is common with duodenal ulcers. Those with gastric abdominal painBloatingAbdominal fullnessNausea and vomitingWeight loss/weight gainHematemesisMelenaWarning symptoms or alarm symptoms or alarm symptoms or alarm symptoms or alarm symptoms of PUD requires history taking physical examination, and invasive/non-invasive medical tests. A careful history should be obtained and noted for the presence of any complications. Patient reporting of epigastric abdominal pain, early satiety, and fullness following a meal raise suspicion of PUD. The pain of gastric ulcers increases 15 to 30 minutes 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. Any patient presenting with anemia, melena, hematemesis, or weight loss should be further investigated for complications of PUD, predominantly bleeding, perforation, or cancer. A physical exam may reveal epigastric abdominal tenderness and signs of anemia. 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 dyspeptic symptoms should be evaluated by an EGD. 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. It may be used to confirm eradication after 4 to 6 weeks of stopping treatment. 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. 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. It is indicated if eradication treatment fails or there is suspicion about antibiotic resistance. Biopsies from at least 4-6 sites are necessary to increase sensitivity. Gastric ulcers are commonly located on the lesser curvature between the antrum and fundus. The majority of duodenal ulcers are located in the first part of the duodenum.

6. Computerized tomography of the abdomen with contrast is of limited value in the diagnosis of the duodenum. PUD itself but is helpful in the diagnosis of its complications like perforation and gastric outlet obstruction. Medical Treatment Antisecretory drugs used for peptic ulcer disease (PUD) include H2-receptor antagonists and proton pump inhibitors (PPIs). PPIs have largely replaced H2 receptor blockers due to their superior healing and efficacy. PPIs block acid production in the stomach, providing relief of symptoms and promoting healing. Treatment may be incorporated with calcium supplements as long-term use of the PPIs can increase the risk of bone fractures. NSAIDs induced PUD can be treated by stopping the use of NSAIDs or switching to a lower dose. Corticosteroids, bisphosphonatesides, bisphosphonatesides, bisphosphonates and promoting the use of the PPIs can increase the risk of bone fractures. and anticoagulants should also be discontinued if possible. Prostaglandin analogs (misoprostol) are sometimes used as prophylaxis for NSAID-induced peptic ulcers. First-line treatment for H. pylori-induced peptic ulcers. First-line treatment for H. are used for 7 to 14 days.[7] Antibiotics and PPIs work synergistically to eradicate H. pylori.[8] The antibiotic resistance in the environment. If first-line therapy with bismuth and different antibiotics is used. 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 refractory peptic ulcer is one over 5 mm in diameter that does not heal despite 8-12 weeks of PPI therapy. The common causes are persistent H. pylori infection, continued use of NSAIDs, or significant comorbidities that impairs ulcer healing or other conditions like gastrinoma or gastric cancer. If the ulcer persists despite addressing the above risk factors, patients can be candidates for surgical treatment. 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. 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 galistones. [10] Elevated serum amylase are useful in the diagnosis. Billary 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, is exacerbated by fatty meals, and is associated with nausea and vomiting. 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 ongoing post-prandial epigastric pain[13] and can be mistaken for peptic ulcer disease. 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. 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. NSAID-induced gastric perforation occurs at a rate of 0.3% per patient per year. 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. Following complications can occur in PUD: Upper gastrointestinal bleedingGastric outlet obstructionPerforationP about potentially injurious agents like nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, alcohol, tobacco, and caffeine. If it is necessary to use NSAIDs, use the lowest possible dose and also consider prophylaxis for patients who use NSAIDs, use the lowest possible dose and also consider prophylaxis for patients who use NSAIDs, aspirin, alcohol, tobacco, and caffeine. If it is necessary to use NSAIDs, aspirin, alcohol, tobacco, and caffeine. Stress reduction counseling can be helpful in some cases. Ulcers are differentiated from erosions, whereas lesions greater than 5 mm in diameter are termed ulcers. COX-2 selective NSAIDs are less likely to cause PUD as COX-2 is not expressed on the gastric mucosa. Therefore, in patients with a history of PUD, COX-2 selective NSAIDs are preferred. A gastrin-producing endocrine tumor causes Zollinger-Ellison syndrome or gastrinoma usually arises from the pancreas or duodenum. It results in multiple ulcers in the duodenum and jejunum. It can be diagnosed by measuring serum gastrin levels. An evidence-based approach to peptic ulcer disease is recommended. PUD is a very common disorder that affects millions of people. When left untreated, it has significant morbidity. 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. Because the presentation of PUD is often vaque, healthcare workers, including nurses, need to be aware of this diagnosis. 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 avoiding consumption of too many NSAIDs. Gastroenterology nurses monitor patients, provide education, and keep the team updated on the patient on medication compliance to obtain symptom relief and a cure. A dietary consult should be sought as there is evidence that obesity may be a trigger factor for peptic ulcer disease. Only through a team approach can the morbidity of peptic ulcer disease be decreased. 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[PMC free article: PMC5997469] [PubMed: 29915630] Disclosure: Talia Malik declares no relevant financial relationships with ineligible companies. Disclosure: Kevin Singh declares no relevant financial relationships with ineligible companies.

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