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A Comprehensive Overview Of Duodenal Ulcers: Pathophysiology, Clinical Features, And Management Strategies"

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Abstract

A duodenal ulcer is a specific type of ulcer that occurs in the lining of the small intestine, particularly in the duodenum, which is the first section of the small intestine immediately after the stomach. This condition is characterized by inflammation and the formation of a sore, which can cause significant abdominal pain. Duodenal ulcers are a common cause of abdominal discomfort, and while they can be quite painful, they often heal within a few weeks with appropriate treatment. Duodenal ulcers fall under the broader category of peptic ulcer disease (PUD). PUD encompasses both duodenal ulcers and gastric ulcers, which are ulcers that form in the lining of the stomach. Both types of ulcers result from a disturbance in the mucosal surface, which is the protective lining of the stomach and duodenum. This disturbance can cause the mucosal surface to erode and form ulcers. The mucosal surface consists of several defensive layers, including pre-epithelial, epithelial, and subepithelial components, all of which work together to protect the underlying tissues from damage. The primary symptom associated with duodenal ulcers is dyspepsia, which is a term for upper abdominal pain or discomfort. However, more severe complications can arise if the ulceration progresses, including gastrointestinal bleeding, obstruction of the digestive tract, the formation of a fistula (an abnormal connection between organs), or even perforation of the stomach or duodenum wall. The presence and severity of symptoms can significantly influence the management and treatment approach for the condition. When diagnosing peptic ulcer disease, it's important to consider a patient's history, especially if they have symptoms of dyspepsia, a history of using nonsteroidal anti-inflammatory drugs (NSAIDs), or a previous diagnosis of infection with Helicobacter pylori (H. pylori). H. pylori is a common bacterial infection that is a major cause of peptic ulcers. Therefore, testing for H. pylori is recommended for all patients with peptic ulcer disease, particularly those with duodena ulcers. NSAIDs, which are commonly prescribed to manage pain and fever, can also contribute to the development of peptic ulcer disease, even with short-term use. This is particularly

relevant for children, who may be given NSAIDs to manage fever. Doctors should consider the potential risks and use these medications cautiously, advising caregivers about the importance of moderation and the potential dangers of over use. In summary, duodenal ulcers are a type of peptic ulcer that causes significant abdominal pain and requires careful diagnosis and treatment. Understanding the underlying causes, including the role of H. pylori infection and the impact of NSAID use, is crucial for effective management and prevention of complications**.**

Introduction:

A duodenal ulcer is an inflammation of the small intestine's lining right above the duodenum (the duodenum). Gastric ulcer refers to an ulcer that develops in the stomach lining.

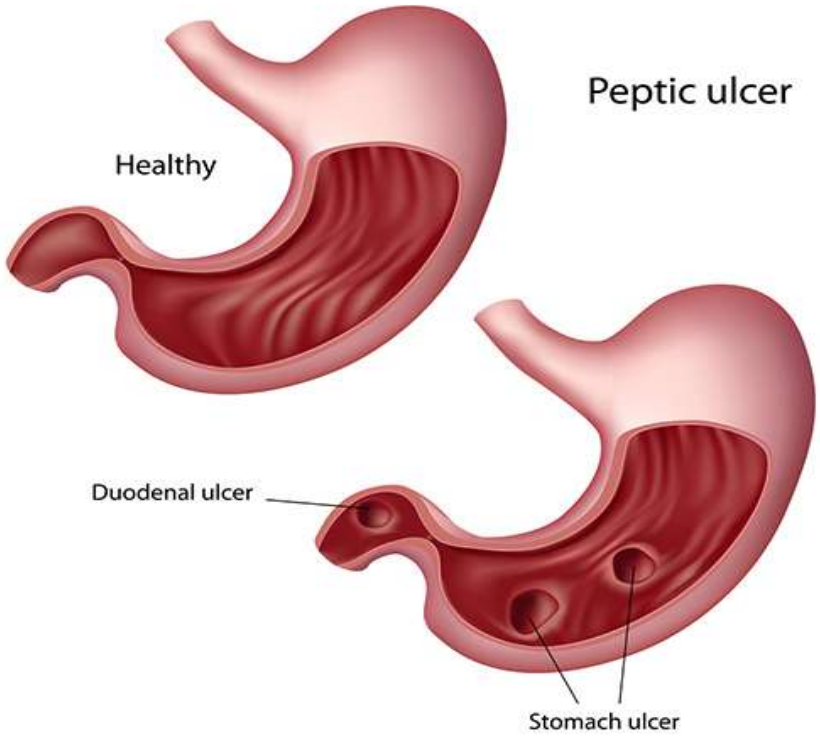
A sore throat that develops in the intestinal lining is called a duodenal ulcer. The first section of your small intestine, or duodenum, is where food passes initially after leaving your stomach in your digestive system. Abdominal pain is frequently brought on by duodenal ulcers. Once treated, they often recover in a few weeks. [1]

Peptic ulcer disease, a more general disease condition, including duodenal ulcers. When there is a disturbance in the mucosal surface at the duodenum, the first segment of the small intestine or the level of the stomach, the condition is referred to as peptic ulcer disease. Anatomically, a defensive system with pre-epithelial, epithelium, and subepithelial components is present on both the gastric and duodenal surfaces Ulceration results from damage to the mucosal surface that extends past the superficial layer. Although dyspepsia is the primary accompanying symptom of most duodenal ulcers, other more severe symptoms might also exist, such as gastrointestinal bleeding, obstruction of the creation of a fistula, a hole, or the gastric outlet. Consequently, the patient's appearance during diagnosis or the disease's course has a significant impact on the management. In persons with symptoms of dyspepsia/upper abdominal pain and NSAID use history or a prior Helicobacter pylori diagnosis, the diagnosis of duodenal vs. gastric ulcer bears examination. Testing for H. pylori should be done on every patient with peptic ulcer disease, and especially those with duodenal ulcers, as this is a common cause.[2]

Causes of Duodenitis Ulcer

Typically, an infection with the bacteria Helicobacter pylori leads to ulcer in the duodenum (H. pylori). After a 4- to 8-week course of acid-suppressing medication, the ulcer will heal. Additionally, the H. pylori infection may often be cured with a one-week course of two antibiotics and an acid-suppressing medication. This typically stops the ulcer from returning. Duodenal ulcers can occasionally be brought on by anti-inflammatory drugs like ibuprofen and aspirin that are used to treat illnesses like arthritis. You might need to take long-term acid-suppressing medication if you need to keep taking the anti-inflammatory prescription. Rarely, other medications or other issues could lead to an ulcer.

Smoking, excessive alcohol consumption, and stress may increase your risk of developing a duodenal ulcer, but these factors are less significant than H. pylori infection.[3]



Symptoms of a duodenal ulcer:

Duodenal ulcers can cause the following symptoms:

- ✓ stomach or abdominal pain.
- ✓ upper abdominal burning indigestion
- ✓ heartburn
- ✓ Feel bloated and extremely full after eating.
- ✓ Feel a bit queasy (nauseous), and lose weight
- ✓ Dark barstools
- ✓ Distension of the abdomen [4]

Pathophysiology of Duodenitis Ulcer

Due to etiological factor

↓

Acid pepsin's increased activity or concentration, as well as the mucosa's lower resistance, both contribute to erosion.

↓

A damaged mucosa is unable to produce enough mucus to serve as a defence against HCl.

↓

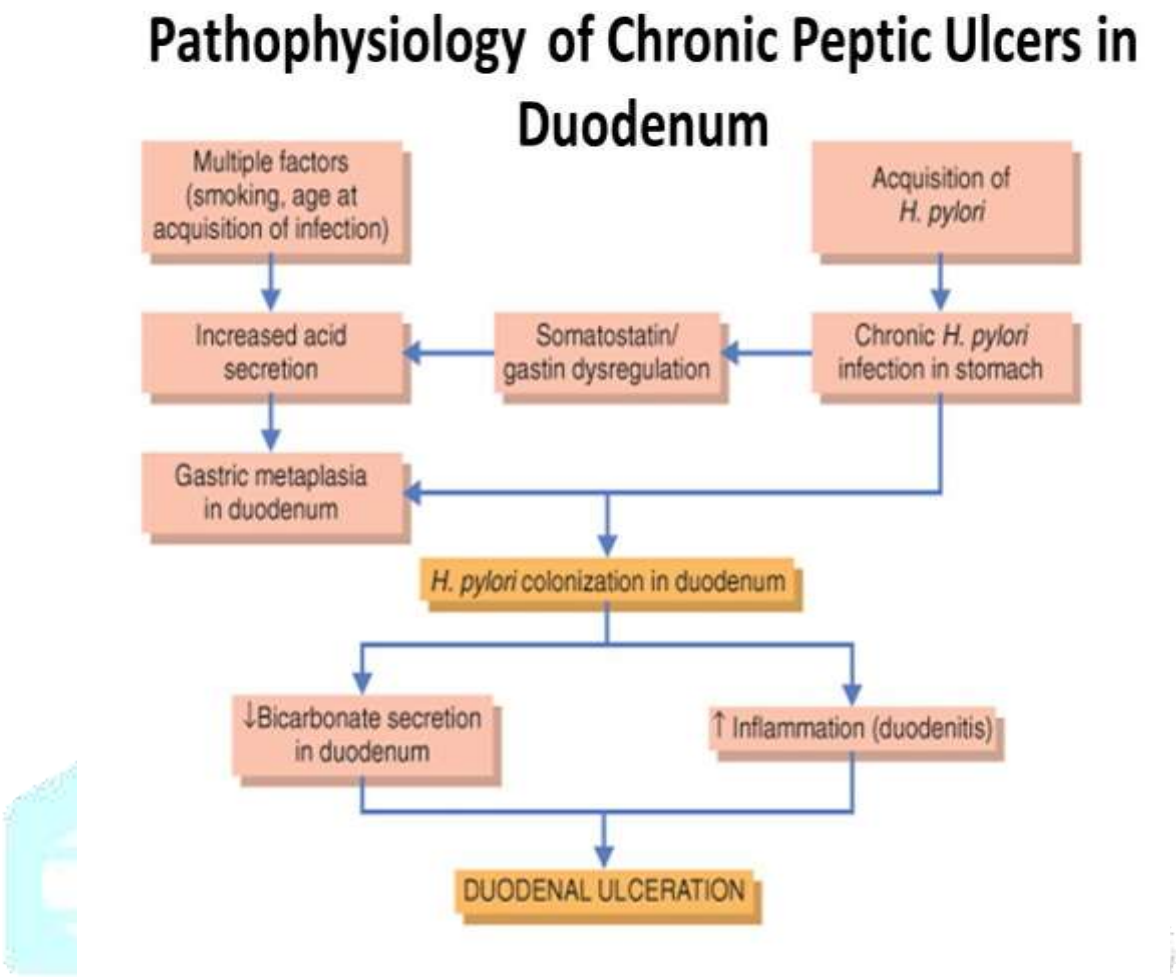
A duodenal ulcer patient produces more acid than usual.

↓

The gastroduodenal mucosa can become damaged, which reduces its ability to fight off pathogens.

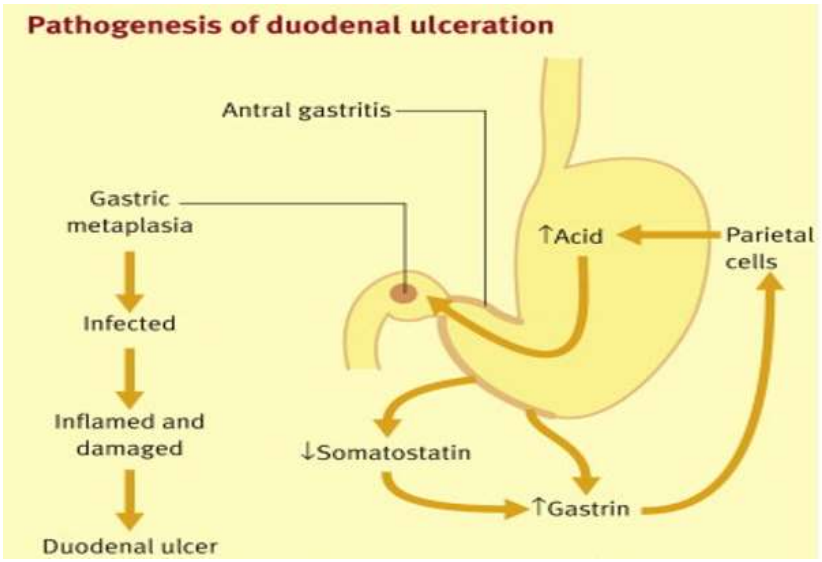
↓

H. Pylori infection arises as a result. [5]



Pathogenesis of Duodenal ulcer

Due to somatostatin's negative feedback impact on gastrin synthesis, antral inflammation reduces somatostatin production, which causes hypergastrinemia. Gastrin causes enterochromaffin-like cells to release histamine, which affects on parietal cells. to cause high-stimulated acid production, an increase in the amount of acid in the duodenum, and the development of protective gastric metaplasia. Helicobacter pylori can colonise gastric metaplasia but not the normal duodenum, which results in inflammation and ulceration..[6]



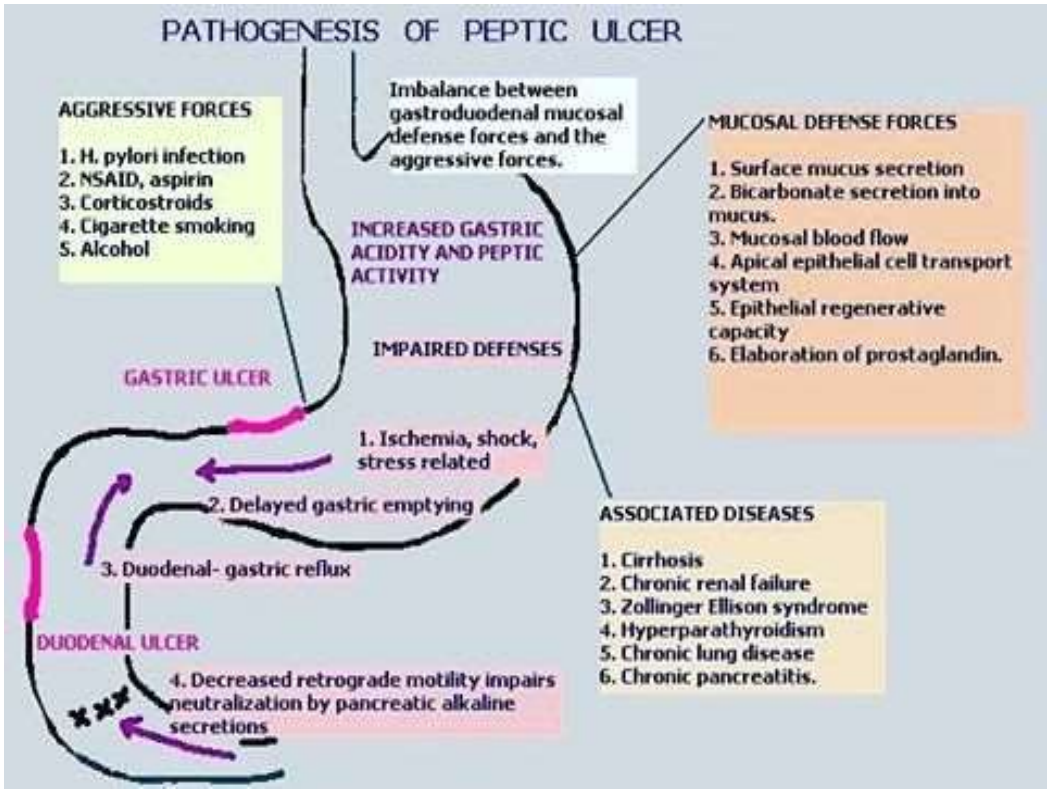
Pathogenesis of H. pylori

Three stages can be used to characterise the pathogenesis of human Helicobacter pylori infection:

1. the ability to enter, adhere to, and colonise the human stomach mucosa;

2 avoidance, manipulation, or abuse of the immune system in humans; and

3 the spread to a new vulnerable host, tissue injury, and proliferation [7]



Treatment of Duodenal Ulcer:

Duodenal ulcers are often treated with a cocktail of drugs that decrease acid production, safeguard the mucosa, and eradicate the H. pylori bacteria (if present).

This promotes ulcer healing and lowers the likelihood of recurrence. Every drug should be taken as directed.[8]

Antiulcer medication

Examples of drugs that could be used to treat duodenal ulcers include:

• H. pylori-killing antibiotics (usually two or three different antibiotics are taken in combination for one to two weeks)

• H2 receptor blockers that reduce gastric acid production (like cimetidine or famotidine)

• Proton pump inhibitors to avoid stomach acid production (such as esomeprazole, lansoprazole, omeprazole, or pantoprazole)

• Surface treatments that hasten healing by protecting the ulcer from enzymes and acid (like sucralfate)

. Bismut (may shield the lining and eliminate bacteria)

• Misoprostol[9]

Antibiotics:

You will be instructed to take two or three antibiotics as well as a PPI if your ulcers are the result of an H. pylori infection. There is solid proof that such eradication therapy, especially for duodenal ulcers, helps heal ulcers and prevent them from coming back. Amoxicillin, clarithromycin, metronidazole, and tetracycline are among the antibiotics that are frequently utilised. They come in a variety of dose forms, including tablets and capsules, and are exclusively prescribed medications. These antibiotics can have moderate side effects, which can include feeling unwell, diarrhoea, and vomiting..[10]

Table No. 1: Some of the antibiotic drugs with their common side effects

Types of drugs (Antibiotics)	Common side effects
Amoxicillin	• Skin rashes • Adverse gastrointestinal reactions such diarrhoea, nausea, and vomiting
Clarithromycin	• Mild gastrointestinal symptoms such nausea, vomiting, stomach pain, and diarrhoea; • Disturbance of smell and taste; • Stomatitis; • Glossitis; • Headache
Metronidazole	• An unpleasant metallic taste; • Gastrointestinal disturbances such as nausea and vomiting; • A tongue that is furred; • Anorexia
Tetracycline	• Dysphagia; • Oesophageal irritation; • Gastrointestinal side symptoms such diarrhoea, nausea, and vomiting • Photosensitivity • Sensitivity responses • The colour of the skin

H2 receptor blockers:

- ✓ Cimetidine
- ✓ Famotidine
- ✓ Nizatidine
- ✓ Ranitidine

By binding to histamine type 2 receptors on the basolateral (anti luminal) surface of gastric parietal cells, H2 receptor blockers prevent the production and release of gastric acid. H2 blockers must be able to selectively block histamine type 1 receptors, which are already inhibited by common antihistamines used to treat allergic reactions but have little impact on the production of stomach acid. The therapeutic indications, side effects, and activity spectra of the four H2 receptor blockers that are easily accessible in the United States are all similar. Since they are so well tolerated, the majority of the general population utilises these medications to treat peptic ulcer disease, heartburn, esophagitis, and various mild upper gastrointestinal symptoms. [11]

Adverse effects:

- In general, H2 receptor antagonists are well tolerated. Headache, drowsiness, exhaustion, abdominal pain, or diarrhoea .
- • In people with renal impairment, hepatic impairment, or who are older than 50, the use of H2RAs has been linked to central nervous system adverse effects such as delirium, disorientation, hallucinations, or slurred speech. Although famotidine has had similar results, cimetidine is typically considered to be the most frequent cause of these symptoms.
- Tachyphylaxis or tolerance may develop with regular usage of H2 receptor antagonists, which limits their ability to be used as a maintenance therapy for GERD symptoms. Within 7 to 14 days of continuing treatment, H2RAs can cause tolerance. H2RAs taken infrequently or as needed may aid in halting the onset of tachyphylaxis..
- H2RAs are less likely than proton pump inhibitors to cause infections and bacterial overgrowth..[12]

Proton pump inhibitors (PPIs):

More than 90% of the generation of stomach acid can be prevented by proton pump inhibitors (PPIs). These medications are more effective at preventing and treating ulcers than antacids or H2 blockers. Although some people may require up to 4 more weeks of treatment to fully recover, the majority of ulcers heal within 4 weeks.

In high risk NSAID-taking patients, proton pump inhibitors (PPIs) including pantoprazole, omeprazole, and lansoprazole have been found to be beneficial in preventing the formation of gastric and duodenal ulcers [13]

Proton pump inhibitors' most frequent adverse effects include the following:

- Diarrhoea
- Constipation
- Pain in abdominal
- Fever
- Vomiting
- Nausea
- Rash

Low levels of magnesium have also been linked to long-term PPI use (hypomagnesemia). A study of patients who used PPIs for a long time revealed an elevated risk of heart attacks. It is crucial to treat the disease at hand with the lowest doses and shortest treatment periods possible.[14]

Additional detrimental side effects of PPIs include:

- Stevens-Johnson syndrome,
- toxic epidermal necrolysis,
- severe allergic reactions, and decreased kidney function

Protectants (Like Sucralfate):

According to your doctor's recommendations, sucralfate is used to treat and prevent duodenal ulcers as well as other diseases. It functions by creating a covering or barrier over the ulcer. This shields the ulcer from the stomach's acid, enabling healing. An aluminium salt is included in sucralfate. An ulcer may not entirely recover for up to eight weeks. Sucralfate neutralises between 14 and 16 mEq of stomach acid per 1g dosage.[15]

Adverse Effects:

Sucralfate works locally and absorbs very little, making it quite safe. Constipation, which affects 1 to 10% of individuals, is the most frequent adverse reaction. When using sucralfate, diabetic people have also experienced hyperglycemia. Other minor adverse effects include stomach bezoar development, headaches, dry mouth, and nausea and vomiting. etc.

Sucralfate interacts with a number of medications and can lower the levels of digoxin, levothyroxine, furosemide, quinolones, oral phosphate supplements, warfarin, and bisphosphonates in the serum, among other medications. Sucralfate administration and the administration of these drugs should be separated by at least two hours.

Sucralfate and aluminium serum concentrations can rise after taking multivitamins. A few drugs, such as antacids, can lessen the effectiveness of sucralfate by reducing its capacity to bind to gastrointestinal ulcers when taken within 15 minutes after sucralfate.[16]

Bismuth:

It is exceedingly misleading to refer to bismuth sub citrate as an antibacterial agent because it is a very powerful antiulcer agent that works independently of its antimicrobial activity. The antiulcer properties of colloidal bismuth sub citrate are not just useful against H. pylori-related ulcers. Duodenal ulcers are treated with tetracycline, metronidazole, and bismuth in addition to conventional ulcer medicines. It belongs to a group of drugs termed antibacterial agents. It functions by halting Helicobacter pylori bacteria development and spread, which frequently happens in ulcers.[17]

Adverse effects:

The effect of bismuth chelate is less likely with tablets, making them more tolerable. Especially in a liquid formulation, bismuth chelate darkens the tongue, teeth, and stool. It is wise to steer clear of the medication in individuals with compromised renal function because although there is little systemic absorption of bismuth from the chelated preparation, it does pass into the urine. Months after bismuth use has stopped, urinary elimination persists. Encephalopathy is brought on by bismuth poisoning..[17]

Misoprostol:

Mechanism of action:

Prostaglandin E1 receptors on parietal cells in the stomach are directly stimulated by the prostaglandin E1 analogue misoprostol suppresses baseline and nocturnal gastric acid output. Through this action, stomach acid secretion that has been stimulated by food, alcohol, NSAIDs, histamine, coffee, etc. is inhibited. There is typically a dose-dependent association with this effect.

Misoprostol promotes edoema of the mucosa and submucosa, mucus and bicarbonate secretion, and mucosal bilayer thickening, which keeps the mucosa's ability to produce new cells by reducing hydrogen ion backflow and improving regulation of mucosal blood flow.. [19]

Adverse effects:

• Shivering/chills, diarrhoea, abdominal discomfort, nausea, vomiting, constipation, dyspepsia, headache, breakthrough bleeding, and irregular menstruation are among the most often reported adverse effects, which are typically minor. Lethargy, weakness, and vertigo are moderate side effects that are less frequently observed.

• Self-limiting diarrhoea, abdominal pain, and side effects are most frequently reported, and these are likely to be related to exposure to the misoprostolic acid released during its metabolism. The discovery that symptom severity frequently correlates with misoprostolic acid's peak plasma concentration serves as the foundation for this line of thinking.

• Uterine rupture is a danger associated with misoprostol use. When misoprostol is used to induce labour in the third trimester, especially when combined with additional risk factors like a prior caesarean section, this risk is typically at its maximum. During a misoprostol-based medical abortion in the first trimester, rupture is uncommon. However, there is a chance for uterine infection to develop later, as with all uterine ruptures.[19]

Case study

Case Report: A 3-year-old child developed peptic ulcer disease after taking nonsteroidal anti-inflammatory medicines for a brief period of time.

Abstract

Background:

In the USA, the UK, and Europe, 1-2 out of every 1000 persons suffer from peptic ulcer disease (PUD), which affects children and adolescents less commonly than it does adults. PUD primarily manifests in youngsters throughout their second development decade. Nonsteroidal anti-inflammatory drugs (NSAIDs), which are routinely prescribed to treat acute febrile illnesses or discomfort in healthy children, are one of the risk factors, however they have only very rarely been associated with PUD and upper gastrointestinal bleeding..

Case presentation:

We discuss an unusual case of upper gastrointestinal haemorrhage in a 3-year-old child that followed a little ibuprofen dose. The patient with a family history of peptic ulcer disease is experiencing fever, coffee grounds vomiting, and stomach pain. ulcers was hospitalised. An altered general health state was evident from the clinical examination, along with a swollen and mildly painful belly that moved normally with breathing and normal stool. Anemia with reticulocytosis was detected in the initial laboratory test. The patient experienced a second episode of vomiting coffee grounds in the first few hours of being in the hospital. An upper digestive endoscopy and biopsy were performed six hours later and revealed a non-bleeding gastric ulcer at 2 cm from the pylorus. The results of the Helicobacter pylori test were poor. Esomeprazole, a proton pump inhibitor, 10 mg daily was administered to the patient for two months. Hemoglobin readings returned to normal, there were no more gastrointestinal complaints, and the follow-up endoscopy revealed that the ulcer had healed.

Conclusion; PUD can result from the use of NSAIDs at the recommended dose, and proper management can result from taking into account the risk factors prior to administration.

Keywords:

paediatrics, nonsteroidal anti-inflammatory medications, upper gastrointestinal haemorrhage, peptic ulcer disease, and proton pump inhibitors.

Case presentation

Having a family history of peptic ulcers, a 3-year-old daughter was admitted with a fever, vomiting coffee grounds, and abdominal pain. Her hemodynamics (128 beats per minute heart rate, 108/71 mmHg blood pressure, and 28 breaths per minute respiration rate) were steady. The patient's mother reported that the patient had taken two doses of paracetamol (250 mg - 16.66 mg/kg by mouth) and two doses of ibuprofen (two doses of 100 mg -6.66 mg/kg by mouth, eight hours apart) to treat a fever within the previous 24 hours.

The patient had a history of upper respiratory tract infections with febrile seizures and interstitial pneumonia treated with antypiretics. For the first three days of the upper respiratory infection (5 weeks before to the bleeding episode), ibuprofen 100 mg, or 6.66 mg/kg, was given orally once every eight hours. The next two days, with a suitable rest in between, ibuprofen was given twice daily. Anti-seizure medications weren't required throughout the convulsion occurrence. Additionally, clarithromycin was ingested orally for 10 days at a dose of 7.5 mg/kg/day. A two-week period was spent with the symptoms.

Amoxicillin/clavulanic acid and cephalosporins are both allergens for the patient. There was no known immune deficiency illness.

During the clinical examination, it was discovered that the patient had a normal breath sound, general malaise, pallor, fever, pharyngotonsillar congestion, a productive cough, a bloated and mildly uncomfortable abdomen that moved normally with breathing, and normal stool. The patient's height and weight were both in the 72nd percentile at 88 cm and 15 kg, respectively (z-scores -1.63 and 0.65). She is at the 98th percentile for her age group with a body mass index (BMI) of 19.4 (z-score 2.15).

Initial laboratory findings included reduced total protein (5.52 g/dL), reticulocytosis, and anaemia (Hematocrit 29.7%, Hemoglobin 9.6 g/dl, reticulocytes 3.6 percent, corrected reticulocyte count 3.24) in addition to reticulocytosis. The results of all other lab tests, including those for coagulation, were normal.

Soon after being taken to the hospital, the patient experienced a second episode of coffee-ground vomiting.

At 2 cm from the pylorus, a non-bleeding stomach ulcer was discovered following an upper digestive endoscopy and biopsy (figure). Gastric biopsy tests for H. pylori came up negative..

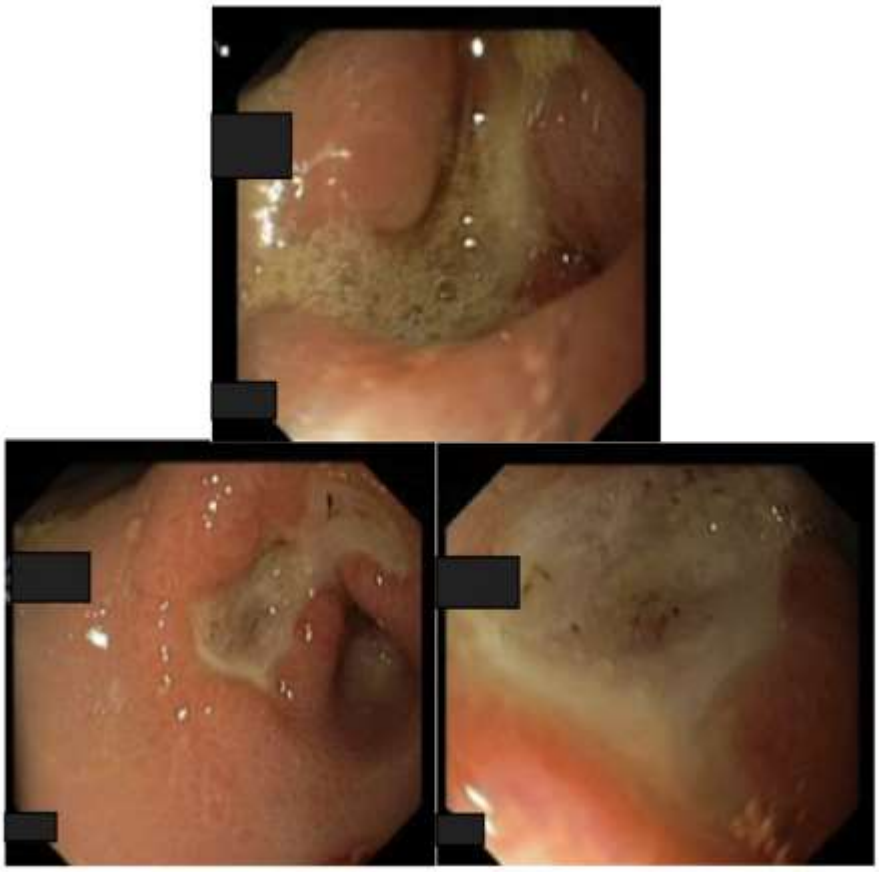


Figure No.1

Based on these findings, it was determined that the upper gastrointestinal bleeding was caused by NSAID-induced stomach ulcer.

During hospitalisation, perfusion with glucose and electrolytes was administered to replenish fluid loss. The patient received a proton pump inhibitor (esomeprazole 10 mg/day - 0.66 mg/kg/day) for two months.

No additional gastrointestinal symptoms appeared. Following the completion of the therapy period, the endoscopic report revealed that the stomach ulceration area had healed as haemoglobin values reverted to normal, indicating cessation of gastrointestinal bleeding.

Discussion:

An upper gastrointestinal haemorrhage in a 3-year-old following brief NSAID use is unusual. Similar examples have been reported in the literature, but it is still unclear how short-term NSAID use in children harms them or how it relates to PUD. However, other studies offer compelling evidence pointing to particular risk factors, such as the age of the kid,NSAID use, and H. pylori infection. [21,25-27]

PUD appears to have the greatest impact on patients between the ages of 10 and 20. According to a retrospective cohort study, people with gastric ulcers tend to be younger than people with duodenal ulcers. [27]

The use of NSAIDs is the second crucial component. The likelihood of PUD rises with treatment length, dose, the presence of risk factors including a positive family history or drug coadministration, and the dose. As a result, despite taking a small amount of ibuprofen, the stomach ulcer (GU) in this case can be partially

attributable to a favourable family history and a connection with a dose of paracetamol. Adults who were taken paracetamol and NSAIDs at a dosage more than 2g have reported experiencing a joint impact from the two medications.[29] On the other hand, there is currently insufficient biological and clinical evidence to support the claim that regular paracetamol use increases the risk of gastrointestinal ulcers and their consequences.

It is well recognised that the population of children with PUD is increasing in other risk factors including a favourable family history, the use of NSAIDs, or H. Pylori infection. The patient's father was found to have PUD, but it was unknown if he also had H. Pylori.

Furthermore, some studies have found a substantial correlation between short-term NSAID use and GU. [25] Studies have connected the use of short-term NSAIDs with proton pump inhibitors (PPIs), which theoretically could reduce the frequency of upper gastrointestinal bleeding in children. Although it is considered safe to coadminister NSAIDs and PPIs to treat unpleasant gastrointestinal symptoms in adults,[30] there is inadequate data on whether this pharmacological association can shield kids from developing short-term NSAIDs-PUDs.

The third important risk factor for PUD, H. pylori infection, wasn't present in our case. Studies suggest that compared to adults, children may have a lesser association between H. pylori and PUD. [21,31] However, it is widely recognised that this infection aggravates chronic gastritis. and is crucial to the development of PUD in kids. [32]

Patients who experience gastrointestinal bleeding due to ulcers brought on by NSAID use should stop using them. Depending on the severity of the presentation, several therapeutic approaches are used. To accomplish hemostasis, There are pharmacologic, endoscopic, and surgical techniques available. Massive bleeding necessitates immediate endoscopic or surgical action. Blood transfusion requirements, lab tests, and systems for classifying paediatric upper gastrointestinal haemorrhage are still being established. [33,34] The endoscopic examination findings of a non-bleeding gastric ulcer and the clinical manifestation of two episodes of isolated hematemesis in the current instance suggested the use of pharmaceutical therapy (coffee-ground vomiting).

Conclusion:

The short-term, appropriate-dose use of NSAIDs, which are routinely given to children to manage fever, can lead to PUD. Risk factors must be considered prior to administration, such as the use of additional antipyretic drugs or a potential family history. Doctors should promote the use of NSAIDs in moderation and warn caregivers about the risks.

Data availability:

There is no need for additional sources because the article has all of the information needed to understand the results.

Permission:

The patient's parents provided their verbal and written agreement in order for this case report to be published..

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