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REVIEW

Gastric pain

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Gastric pain may be generalised, diffused, specific to the right or left upper quadrant (or both), and may be attributed to a range of possible causes. Types of gastric pain include dyspepsia and epigastric pain. The term "gastric pain" is not frequently encountered in the literature. Therefore, the main focus of this review is on epigastric pain and dyspepsia, both of which are frequently encountered in the clinical setting. For example, it is estimated that dyspepsia affects a quarter of the global population. Several drugs and drug classes are also linked to a range of mechanisms through which the drugs induce mucosal injury in the upper gastrointestinal tract. Therefore, this article provides an overview of the aetiology, classification, risk factors, diagnostic criteria and management strategies aimed at gastric pain, and its two more distinct gastrointestinal-related manifestations, namely epigastric pain and dyspepsia.

Keywords: gastric pain, epigastric pain, dyspepsia, peptic ulcer disease (PUD), GORD, proton-pump inhibitors

Introduction

The term "gastric pain" originates from the Greek word, *gaster,* and modern Latin term, *gastrics,* used in the mid-17th century, which translates to "of the stomach". Gastric pain is currently commonly used to describe pain or discomfort in the upper abdomen. It may be generalised, diffused, specific to the right or left upper quadrant (or both), and may be as a result of a range of possible causes. Types of gastric pain include dyspepsia and epigastric pain. Epigastric pain is defined as pain that is localised in the upper-middle region of the abdomen.[1-3]

Conversely, "dyspepsia" is the often recurring sensation of either pain or discomfort in the area of the upper abdomen. The term is used to refer to a variety of symptoms which are thought to originate in the gastrointestinal tract. These include heartburn, epigastric pain, nausea, vomiting, a feeling of early fullness (satiety) and bloating.[4] Dyspepsia is a complex disease. Numerous potential mechanisms underlie its pathophysiology, including abnormal intestinal motility and visceral hypersensitivity, as well as genetic, infectious, post-infectious and psychosocial factors.[5]

The term "gastric pain" is not frequently encountered in the literature. Therefore, the main focus of this review will be on epigastric pain and dyspepsia.

Aetiology

Epigastric pain has several different causes which are linked to the localisation of gastric pain. Table 1 provides an exposition of such causes, grouped according to the area of discomfort.[6]

Table 1: Different areas of gastric pain and their associated causes[6]

Area	Possible causes
General gastric pain	• Stomach ulcers • Heartburn and indigestion • Pancreatitis • Epigastric hernia • Gallstones
Diffuse gastric pain	• Acute pancreatitis • Diabetic ketoacidosis • Early appendicitis • Gastroenteritis • Intestinal obstruction • Mesenteric ischaemia • Peritonitis (any cause) • Spontaneous peritonitis • Typhoid fever • Sickle cell crisis
Right or left upper quadrant pain	• Acute pancreatitis • Herpes zoster • Lower lobe pneumonia • Myocardial ischaemia • Radiculitis
Right upper quadrant pain	• Cholecystitis and biliary colic • Congestive hepatomegaly • Hepatitis or hepatic abscess • Perforated duodenal ulcer • Retrocecal appendicitis (rarely)
Left upper quadrant pain	• Gastritis • Splenic disorders (abscess and rupture)

Risk factors for the development of epigastric pain and dyspepsia

The following factors increase the likelihood of significant organic disease, and are trigger points for referral:[7]

- *Advancing age:* Being 50 years of age or older at the first presentation. (The incidence of gastric cancer increases with age)
- A family history of gastric cancer, especially when the age of onset is younger than 50 years
- Severe or persistent dyspepsia
- Treatment failure
- A history of peptic ulcer disease, particularly if complicated
- The consumption of nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin
- Severe, debilitating pain, or pain which wakes the patient at night
- Referred pain
- Chronic gastrointestinal tract bleeding signs and symptoms, including melena stools
- Iron-deficiency anaemia
- Dysphagia
- Persistent or protracted vomiting, with or without blood, or the persistent regurgitation of food
- A palpable abdominal mass
- Coughing spells or nocturnal aspiration
- Unexplained weight loss
- Continued changes in bowel habits.

Criteria for the diagnosis of epigastric pain and dyspepsia

Because dyspepsia is a frequent clinical problem, the cornerstone of the initial evaluation rests upon the identification of possible causes. Heartburn and retrosternal pain are exceptions to the diagnostic criteria for dyspepsia. Heartburn is believed to emerge from the oesophagus, which may be more indicative of gastro-oesophageal reflux disease (GORD). However, it may still occur as a coexistent condition. Conversely, non-cardiac chest pain should be differentiated from retrosternal pain, which originates from the oesophagus.[8]

Furthermore, as per the Rome III criteria, symptoms are typically present for at least three months (symptoms may also classically be sustained over six months or longer) in the case of functional dyspepsia. Dyspepsia is also grouped by the Rome III criteria into two separate categories, namely postprandial distress syndrome (PDS) and epigastric pain syndrome (EPS) (Table 2).[8] In addition, the common causes of dyspepsia and their suggestive findings are outlined in Table 3.

A number of less probable causes should also be considered, including the following:[8]

- Pancreatic or hepatobiliary tract disease
- Coeliac disease
- Infiltrative diseases of the stomach
- Motility disorders
- Metabolic disturbances
- Intestinal angina

Table 2: Diagnostic criteria for postprandial distress syndrome and epigastric pain syndrome[8]

Postprandial distress syndrome	Epigastric pain syndrome
Troublesome postprandial fullness, after a standard-sized meal, occurring several times per week (at a minimum) **And/or** Early fullness which prevents a regular meal from being finished, occurring several times per week (at a minimum)	Pain or burning localised to the epigastrium of at least moderate severity at a minimum of once per week **and** Intermittent pain **and** Not generalised or localised to other abdominal or chest regions **and** Not relieved by defecation or passage of flatus **and** Not fulfilling the criteria for disorders of the gallbladder and/or sphincter of Oddi
Supportive criteria	
• Upper abdominal bloating, postprandial nausea or excessive belching • Epigastric pain syndrome may be coexistent	• The pain may be of a burning quality, but without a retrosternal component • The pain is commonly induced or relieved by the ingestion of a meal, but may occur during fasting • Postprandial distress syndrome may be coexistent

- Bacterial overgrowth of the small intestine

Table 3: Common causes of dyspepsia and their suggestive findings[9]

Cause	Suggestive findings
Achalasia	• Slowly progressive dysphagia • Early satiety, nausea, vomiting and bloating • Symptoms are exacerbated by food • Occasional nocturnal regurgitation of the undigested food • Chest discomfort
Cancer, i.e. oesophageal or gastric	• Chronic or vague discomfort • Later, dysphagia (oesophageal) or early fullness (gastric) • Weight loss
Coronary ischaemia	• Symptoms described as gas or indigestion, rather than chest pain by some patients • May have an exertional component • Cardiac risk factors
Delayed gastric emptying, caused by diabetes, viral illness or drugs	Nausea, bloating and fullness
Oesophageal spasm	Substernal chest pain, with or without dysphagia for liquids and solids
Gastro-oesophageal reflux disease	• Heartburn • Occasional reflux of acid or the stomach contents into the mouth • Symptoms occasionally triggered by lying down • Relief with antacids
Peptic ulcer disease	Burning or distressing pain, relieved by food or antacids

Dyspepsia causes

The majority of cases constitute functional dyspepsia

Five major causes include:
- GORD
- Chronic peptic ulcer disease
- Gastric cancer and other malignancies
- Gallstones
- Medication
- Functional dyspepsia

Dyspepsia risk factors

The risk factors for dyspepsia are as follows:

- A history of previous peptic ulcer disease
- Age > 60 years
- High-dosage or prolonged use of NSAIDs
- Peptic ulcer disease that is associated with *Helicobacter pylori*
- Chronic alcohol use
- A smoking habit
- Stress and depression

Dyspepsia characteristics

The characteristics of dyspepsia are as follows:

- A feeling of postprandial fullness or heaviness
- Early satiation
- Acid heartburn (associated with GORD), epigastric pain and epigastric burning
- Non-cardiac chest pain
- Less specific symptoms include nausea, vomiting, bloating, belching, gas and abdominal distension

(Patients who suffer from functional dyspepsia usually experience intermittent symptoms over the long term, interspersed with periods of remission)

GORD: gastro-oesophageal reflux disease, NSAIDs: nonsteroidal anti-inflammatory drugs

Figure 1: Common risk factors, causes and characteristics of dyspepsia[8,10-13]

- Irritable bowel syndrome
- Diabetic radiculopathy
- Intestinal hernia
- Abdominal wall pain.

Classification of dyspepsia

Dyspepsia can be classified as being either organic or non-organic in nature.[9-11]

Organic dyspepsia has a known underlying cause, such as gastric cancer, peptic ulcer disease (PUD), chronic alcohol use and drug-induced gastric discomfort, or stress. Treatment of the underlying cause may eliminate the symptoms.

Non-organic (functional or non-ulcer) dyspepsia is characterised by the presence of dyspeptic symptoms originating in the gastroduodenal region, but with no indicated abnormalities on physical examination and upper gatrointestinal endoscopy.

Figure 1 summarises the most common risk factors, causes and characteristics of dyspepsia.

Globally, approximately 25% of the population suffers from dyspepsia, with rates ranging from 13–40% in different countries.[14]

Red flags for dyspepsia

According to Greenberger, the following findings are of particular concern:[9]

- Acute episodes accompanied by dyspnoea, diaphoresis or tachycardia
- Anorexia
- Nausea or vomiting
- Weight loss
- Blood in the stools
- Dysphagia or odynophagia
- Failure to respond to therapy with H_2-receptor blockers or proton-pump inhibitors (PPIs).

If a patient complains of indigestion, the healthcare professional should contemplate all potential causative factors, including those originating from the oesophagus, stomach, heart, liver, gall bladder, pancreas and intestines, as well as the use of NSAIDs and other medication.[15]

Medication which may cause dyspepsia and epigastric pain

Medicine is commonly seen as a potential causative factor of dyspepsia. However, the symptoms experienced are often assigned to a disease, rather than to drug therapy. Owing to the high occurrence of dyspepsia, it is troublesome to differentiate between spontaneous and true drug-related dyspepsia.[4]

The sensitivity of the patient is a considerable contributing factor to the experienced symptoms. Other contributing factors include dose-related side-effects, a history of pre-existing disease, e.g. a peptic ulcer, and the intrinsic defects of the gastrointestinal tract, e.g. gastric atrophy, coeliac disease and ageing.[4]

Individual mechanisms are of significance in determining side-effect profiles and accompanying individual susceptibility factors. These include age, gender, genetic and physiological factors, and underlying disease.[4] Table 4 provides a list of medication which could cause dyspepsia and epigastric pain, together with the associated mechanisms of mucosal injury.

Management strategies for gastric pain

The management of peptic ulcer disease

The treatment of chronic PUD varies, depending on the aetiology of the ulcer (*Helicobacter pylori* or NSAIDs), whether the ulcer is initial or recurrent, and whether complications have occurred. The treatment goal is to relieve pain, heal the ulcer, and prevent ulcer recurrence and to reduce ulcer-related complications.[17]

The goal is to eradicate the organism, to heal the ulcer and to cure the disease for *H. pylori*-positive patients, who should be initiated on a PPI-based, three-drug regimen which includes the PPI once or twice a day, clarithromycin 500 mg twice daily,

Table 4: Medication which could cause dyspepsia and epigastric pain[4,16]

Medication	Mechanism of mucosal injury
Sustained-release medication and hygroscopic medication	
Sustained-release medication and hygroscopic medication	• *Non-specific, direct mucosal injury:* Oesophageal injury can result from direct, prolonged mucosal contact with tablets or capsules due to insufficient clearance from the oesophagus in the supine position. The most common reason is ingestion with an inadequate (< 100 ml) amount of fluid being consumed • Contributing factors include acidity, alkalinity, the drug dissociation rate, osmolality, and intrinsic chemical toxicity and hygroscopicity, which may result in a high ulcerogenic concentration, e.g. potassium chloride • Sustained-release drugs may be more harmful to the oesophagus in the case of prolonged contact
Pain and fever, and anti-inflammatory drugs Examples of NSAIDs are: • Indomethacin • Meclofenamate • Piroxicam	These agents are gastric irritants which may cause damage throughout the gut. The major underlying mechanism of injury is thought to be due to direct cellular toxicity and disruption of the mucosal barrier (a local effect), rather than by inhibiting prostaglandin synthesis (systemic effect)
Acetylsalicylic acid	More likely to cause gastric irritation
COX-2 inhibitors	Cause less damage to the upper gastrointestinal mucosa than traditional NSAIDs, but dyspepsia remains a significant problem
Sulphasalazine	An intestinal anti-inflammatory agent, which may also cause dyspepsia
Corticosteroids	• Dyspepsia occurs with all corticosteroids and is dose related to some extent, but is still dependent on individual sensitivity, and is more common in patients with a history of previous ulcers • Large dosages of glucocorticosteroids have been associated with the development of peptic ulcers due to the suppression of the local immune response to *Helicobacter pylori*
Antimicrobial agents and drugs that act on the gastrointestinal tract	
Macrolides, such as erythromycin, PPIs and H_2-receptor antagonists, share similar mechanisms of injury	• Erythromycin directly stimulates the motilin receptors on the gastrointestinal smooth muscle, which may lead to alterations in gastric motility • An induction of gastric acid rebound induces an increased (and paradoxical) capacity for gastric acid secretion. The suggested mechanism of injury includes changes in gastrin secretion, which is induced by pH changes in the gastric antrum due to prolonged acid inhibition • Gastrin has a confirmed trophic effect on the oxyntic mucosa, which may induce hyperplasia or hypertrophy of the enterochromaffin-like cells and the parietal cells. This rebound acid hypersecretion induces dyspeptic and reflux symptoms in patients once the drugs which inhibit their acid secretion are stopped • Newer macrolides, such as azithromycin, clarithromycin and roxithromycin, are better tolerated and cause fewer adverse events
Tetracyclines	• Mild gastrointestinal disturbances are common. Nausea, vomiting and epigastric burning are the most frequent, but oesophageal ulcers may be the most dramatic • An acute onset of a substernal burning pain and dysphagia has been described in cases where remaining parts of ingested tetracycline capsules have been identified by endoscopy
Itraconazole, terbinafine, ribavirin, abacavir and oseltamivir	Cause gastrointestinal upset and may provoke dyspepsia
Drugs that act on the central nervous system	
Risperidone and the SSRIs	Increased serotonergic activity in the gut is commonly associated with gastrointestinal upset, and may lead to the induction of dyspepsia. However, adverse gastrointestinal effects tend to emerge early on in treatment, and subsequently tend to improve after the first week of therapy
Drugs that act on the immune system	
Oral methotrexate, tacrolimus, D-penicillamine and mycophenolate sodium	Mucositis is a frequent adverse effect of the immunosuppressive agents, and this leads to painful inflammation and ulceration of the mucous membranes that line the digestive tract
Various other drugs	
Iron	• Dyspepsia can be seen with higher dosages • Iron has a well-known reputation for poor gastric tolerance
Oral nicotinic acid, sildenafil and tadalafil, theophylline and the calcium-channel blockers	May provoke GORD owing to the anticholinergic properties of these agents which facilitate the relaxation of the lower oesophageal sphincter
Bisphosphonates	Generally associated with improper ingestion and inappropriate timing of the drug intake
Potassium chloride	Is irritating to the gastrointestinal tract, which may lead to perforations of the gut and finally result in ulcer formation

COX-2: cyclo-oxygenase-2, GORD: gastro-oesophageal reflux, H_2-receptor antagonists: histamine type 2-receptor antagonists, NSAIDs: nonsteroidal anti-inflammatory drugs, PPIs: proton-pump inhibitors, SSRIs: selective serotonin reuptake inhibitors

and amoxicillin 1 g twice daily (or metronidazole twice daily, if allergic to penicillin)[17] (Table 5).[18]

Table 5: Recommended seven-day regimen for the eradication of *Helicobacter pylori* [18]

Rx:
A proton-pump inhibitor, e.g. omeprazole 20 mg, twice daily, for 7 days
or
An H_2-receptor antagonist, e.g. ranitidine 150 mg, twice daily, or 300 mg at bedtime if proton-pump inhibitors are contraindicated
plus
Two of the following antibiotics: • Clarithromycin 500 mg twice daily • Amoxicillin 1 g twice daily • Metronidazole 400 mg twice daily

Rx: treatment

Healing the ulcer as rapidly as possible is the specific goal of patients with NSAID induced-ulcers. Patients who are at risk of developing NSAID-induced ulcers should receive prophylactic co-therapy or use a cyclo-oxygenase-2 (COX-2) inhibitor. PPI co-therapy reduces the risk of NSAID-associated ulcers, is as effective as recommended dosages of misoprostol, and is superior to H_2-receptor antagonists. Standard PPI dosages and nonselective NSAIDs are as effective as selective COX-2 inhibitors in reducing risk.[17]

The intravenous administration of a PPI loading dosage, followed by 72-hour continuous infusion, is the recommended treatment for severe peptic ulcer bleeding, after appropriate endoscopy treatment. The goal is to maintain an intragastric pH of > 6.[17]

The management of dyspepsia

As already mentioned, the treatment approach to patients with dyspeptic symptoms, as for acid heartburn and GORD, aims to[19,20] decrease the amount of stomach acid which enters the distal oesophagus, usually by neutralising the stomach acid, decreasing the production of hydrogen choloride (HCl), and increasing the rate at which the stomach empties into the duodenum, as well as relieving the discomfort caused by heartburn.

The major drug targets in the current practice setting are the so-called proton pump (or the H^+/K^+-ATPase pump), the gastric H_2 receptor and the gastrointestinal 5-hydroxytryptamine 4 (5-HT_4) receptor. These targets may be supported by simple antacids and the prostaglandin analogues. The pharmacotherapeutic measures may be supported by basic, nonpharmacological intervention strategies.

Treatment is determined by the disease severity and includes:[21]

- Lifestyle changes and patient-directed therapy with an antacid, non-prescription H_2-receptor antagonist and/or non-prescription PPI
- Pharmacological treatment with prescription-strength acid-suppression therapy
- Anti-reflux surgery (a viable treatment alternative for patients when long-term pharmacological treatment is undesirable, or when patients have refractory symptoms or complications).

Acid suppression is the mainstay of therapy. The PPIs provide the greatest symptom relief and the highest healing rates, especially in patients with complications, moderate to severe symptoms, or erosive disease.[21]

Nonpharmacological measures

Patients should be advised to refrain from indulging in foods which trigger the onset of dyspeptic symptoms, such as chillies, spices, fat, orange juice, tomato juice, coffee and alcohol. Smaller meals should be taken more frequently, so as to avoid unnecessary gastric distension. Patients are advised to avoid the use of NSAIDs and other medication with a strong link to the occurrence of dyspepsia, wherever possible. If a NSAID must be used, then the patient should also be given preventative therapy to avoid the uncomfortable dyspeptic symptoms.[8,11,21,22]

Additional measures include:[21]

- Elevating the upper body when lying in bed or sleeping at night, to facilitate oesophageal clearance
- Weight reduction in obese patients (reduces symptoms)
- Additional dietary measures include avoiding foods which decrease lower oesophageal sphincter pressure or increase transient lower oesophageal sphincter relaxation, e.g. fats, chocolate, alcohol, peppermint and spearmint, and including protein-rich meals in the diet to augment lower oesophageal sphincter pressure.

Behavioural changes that may reduce oesophageal acid exposure include:[21]

- Eating smaller meals more frequently, and avoiding sleeping immediately after a meal
- Smoking cessation, if applicable
- Avoiding wearing tight-fitting clothes
- Always taking medication while in an upright or sitting position, and with a sufficient quantity of liquid.

Pharmacotherapy

The following drug options may be used to treat dyspepsia.

Simple antacids: Simple antacids, although very widely used in the over-the-counter setting as symptom alleviators for acid heartburn, may have very limited value in the management of functional dyspepsia. Simple antacids, such as those containing aluminium and magnesium, neutralise hydrochloric acid (HCl) in the stomach and are quite effective as pain relievers. The magnesium-containing antacids cause diarrhoea, while the aluminium-containing ones cause constipation. Therefore, a combination of magnesium and aluminium constitutes the antacid of choice, e.g. a combination of aluminium hydroxide and magnesium trisilicate.

However, the divalent cations, i.e. Al^{2+} and Mg^{2+}, interact with chelating agents, such as the tetracycline and fluoroquinolone antimicrobial agents, and several other drug interactions are possible. Combining an antacid with an alginate may actually prevent reflux in that the alginate literally forms a "floating gel" on top of the gastric contents. Calcium carbonate and sodium

bicarbonate may also be used as a simple antacid. However, care should be taken with these agents since calcium carbonate may interfere with the normal acid base balance and cause metabolic alkalosis, or may elicit rebound gastric acid secretion, making it suitable for short-term use only. Sodium bicarbonate should be used with caution in patients who require a restricted sodium intake. Dimethicone and simethicone may relieve a "bloated feeling" by acting as an antiflatulent or defoaming agent. These latter agents may also be of benefit in the management of intestinal colic in infants and children.[19,20,23]

The H_2-receptor antagonists: Blocking the gastric H_2 receptors of the parietal cells reduces stomach acid secretion. Ulcer healing rates are significant, but not nearly as good as those obtained through the use of PPIs, although these agents are good alternatives to PPIs in situations when the latter agents are contraindicated. Cimetidine, ranitidine, famotidine and nizatidine are examples of selective H_2-receptor blockers. Cimetidine has the disadvantage of sometimes producing unwanted anti-androgenic side-effects in male patients. (It has a fairly small affinity for androgen receptors). There is also a higher likelihood of multiple drug interactions through its inhibition of the cytochrome P450 isozymes.[11,19,20,23]

Proton-pump inhibitors: PPIs enter the parietal cells of the gastric glands found in the gastric pits of the stomach lining, where they subsequently inhibit the H^+/K^+-ATPase pump, i.e. the "proton pump" that is specifically responsible for H^+ secretion in the lumen of the gastric pits where these cations combine with the secreted Cl^- from a separate pump to form HCl. This effectively prevents the secretion of gastric acid from the gastric pits into the lumen of the stomach. Therefore, these drugs are highly effective in increasing the stomach pH, rapidly relieving the symptoms and achieving good cure rates. Well-known examples of PPIs are omeprazole, esomeprazole (the *S*-isomer of omeprazole), lansoprazole, pantoprazole and rabeprazole. The PPIs are widely regarded as the first-line treatment option of choice and have well-documented, superior efficacy levels compared to the other acid-lowering drugs that are currently available. PPIs should preferably be taken 30-60 minutes before the first meal of the day for optimal effectiveness.[11,19,20,23]

Cytoprotective agents: These drugs are referred to as cytoprotective because they protect the cells of the stomach lining against the corrosive effects of stomach acid. In addition, misoprostol also promotes the perfusion of the gastric mucosa because it is an analogue of prostaglandin E_1 (PGE_1). These agents may be more effective in the management of acid heartburn than in that of functional dyspepsia. Sucralphate forms a protective layer that covers the exposed surface of the ulcer, and in so doing produces cure rates that are comparable to those obtained with the H_2-receptor antagonists. Preferably, sucralphate should be taken one hour before meals since it is activated by stomach acid. Wherever sucralphate is combined with any of the simple antacids, the antacid should be taken half an hour after taking the sucralphate, i.e. on an empty stomach as well. Misoprostol is of particular use in preventing the gastrotoxic effects of NSAIDs. It influences the ratio of acid-to-mucus secretion favourably by increasing gastric mucus secretion, while decreasing acid secretion. However, care should be taken with this drug since PGE_1 causes uterine contractions, may be used for the termination of pregnancy or the induction of labour, and therefore should be avoided during pregnancy. Bismuth compounds may also be used.[19,20,23]

Prokinetic agents: Metoclopramide acts as an agonist at the gastrointestinal 5-HT_4 receptors, thus increasing the rate of gastric emptying and peristalsis. Domperidone has a similar mechanism of action, but differs from metoclopramide in that it does not cross the blood-brain barrier. Cisapride is another 5-HT_4 receptor agonist, and is unrelated to the previously mentioned two drugs. It has the disadvantage of causing potentially serious cardiac side-effects, such as ventricular dysrhythmias by causing QT_c interval prolongation, especially when its own metabolism is inhibited, for instance, through various drug interactions. Access to this drug has been restricted, and it should be used with extreme caution. Bethanechol is a parasympathomimetic drug which selectively stimulates the muscarinic receptors. This causes smooth muscle contraction in the gastrointestinal tract, but produces relaxation of the sphincters. Therefore, bethanechol stimulates the functional contraction of the gastrointestinal tract, i.e. it increases intestinal motility. The use of neostigmine is a different approach and results in a similar outcome with respect to the motility of the gastroinstesinal tract. Erythromycin also has prokinetic properties.[19,20,23]

Helicobacter pylori *eradication*: Similar to the management of PUD, the eradication of *H. pylori* may be warranted in certain situations, or when a seeming case of functional dyspepsia has not yet been fully evaluated. *H. pylori* damages the mucous lining of the stomach and duodenum. This then exposes the gastric tissue to damage by gastric acid and pepsin, and gastric ulceration occurs. A seven-day regimen is used to eradicate *H. pylori* in patients suffering from dyspepsia in South Africa (Table 5).[18]

Conclusion

Epigastric pain and dyspepsia are two commonly occurring types of gastric pain. It is most likely that dyspepsia originates from the gastrointestinal tract and encompasses a variety of symptoms. These include heartburn, epigastric pain, nausea, vomiting, a feeling of early satiety and bloating. Dyspepsia may also be classified as being either functional or organic in nature, and may manifest itself as either PDS or EPS according to the Rome III criteria. Conversely, epigastric pain, albeit one of the possible symptoms of dyspepsia, may also occur as a symptom of several other conditions of the gastrointestinal system.

Dyspepsia has no known, definitive, underlying patho-physiological mechanism. It is a set of disorders that may even relate to co-morbidities such as malignancies and irritable bowel syndrome. NSAID therapy is another well-known cause. Physicians and healthcare professionals should be aware of dyspepsia and its treatment strategies since it constitutes a significant disease burden worldwide. Effective diagnosis and

determination of the underlying causes of dyspepsia can be labelled as the cornerstone of effective dyspepsia treatment.

Therefore, the need for a differential diagnosis in the clinical setting is significant and should aim to rule out non-gastrointestinal causes of gastric pain, including coronary ischaemia, for example. In addition, several drugs and drug classes are linked to diverse mechanisms of mucosal injury, and hence could elicit dyspepsia and epigastric pain. Thus, a thorough assessment of the patient is required, and must include risk factors, current pharmacotherapeutical interventions, the past medical history, co-morbid conditions and symptomatology.

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