Gastro-oesophageal reflux disease: a pharmacist's perspective for 2020

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Abstract

Gastro-oesophageal reflux disease (GORD) produces symptoms that cause great irritation and discomfort to the patient. Pharmacotherapeutic management is directed at minimising these symptoms and reducing the causative factors, e.g. acid production, thereby providing the necessary relief. Currently available agents include simple antacids and acid suppression therapy, including histamine-2 receptor antagonists, proton-pump inhibitors, mucosal or cytoprotective agents and pro-motility agents. Deciding on appropriate therapy will be dependent upon the diagnosis, side-effects and cost-effectiveness of the treatment.

Keywords: GORD, gastro-oesophageal reflux disease, PPIs, proton-pump inhibitors, histamine-2 receptor antagonists, cytoprotective agents

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Introduction

Gastro-oesophageal reflux disease (GORD) is a medical disorder known and experienced by many, where the gastric contents of the stomach reflux into the distal part of the oesophagus. The incidence amongst members of the Western population is as high as 40%. The exact prevalence of GORD in South Africa is undetermined. Heartburn (dyspepsia) and/or acid regurgitation are common symptoms.[1] GORD may be aggravated by various comorbidities and risk factors. GORD may be classified into three distinct classes, namely:[2-4]

- physiological gastro-oesophageal reflux,
- pathological gastro-oesophageal reflux, and
- secondary oesophageal reflux.

The medical management of GORD is aimed at decreasing the amount of stomach acid that enters the distal oesophagus, usually by increasing the rate at which the stomach empties into

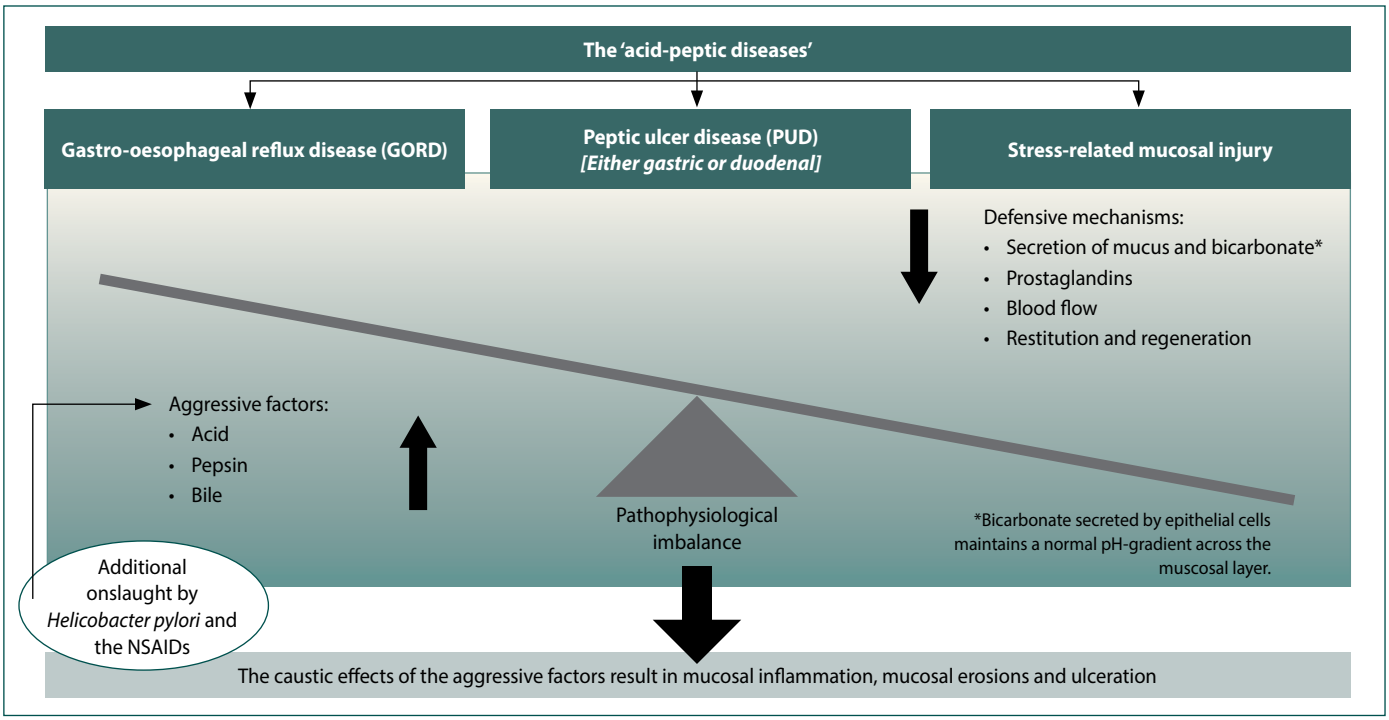

Figure 1: The so-called acid-peptic diseases and their resultant mucosal injury (common contributors to the pathological imbalance between the aggressive factors and the normal defence mechanisms) are infections with *Helicobacter pylori* and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). The latter two aggravating factors contribute to nine in 10 cases of peptic ulceration.[2]

the duodenum, and relieving the discomfort caused by heartburn. From a treatment perspective, however, the distinction between the management of GORD and peptic ulceration is purely arbitrary. Both are peptic acid-related diseases that are characterised by inflammatory and erosive changes in the normal gut mucosa. Both require an essentially similar pharmacotherapeutic treatment approach (refer to Figure 1).[2-4]

Epidemiology

GORD is not necessarily an age-specific condition, but mainly occurs in people older than 40 years of age. The prevalence of GORD varies with the highest incidences being recorded in the Western world. Mortality is rare but can impact patients' economic status and their quality of life. Gender only plays a significant role in the development of Barrett's oesophagus, but not in the case of GORD. In addition, the relationship, and shared risk factors, between recurrent symptoms of GORD and adenocarcinoma of the stomach are yet to be fully elucidated. Recent research has found that patients suffering from major depressive disorder (MDD) show an increasing prevalence of GORD, with particular reference to a study done in Taiwanese patients, amongst others. Risk factors and comorbidities that may worsen or even contribute to the pathophysiology of GORD, include the following:[5]

- family history,
- obesity, with greater chances of GORD,
- smoking,
- alcohol consumption,
- certain medication and foods,
- respiratory diseases,
- reflux chest pain syndrome, and
- major depressive disorder

Pathophysiology

GORD develops when there is abnormal reflux of gastric contents from the stomach into the oesophagus. A defective lower oesophageal sphincter pressure (LOSP) is the main underlying pathophysiological mechanism. Other normal mucosal defence mechanisms contributing to GORD include abnormal oesophageal anatomy, improper oesophageal clearance of gastric fluids, reduced mucosal resistance to acid, delayed or ineffective gastric emptying, inadequate production of epidermal growth factor, and reduced salivary buffering of acid. Oesophageal damage may also be the result of substances like pepsin, bile acids and pancreatic enzymes forming a part of the expelled stomach content (also refer to Figure 1).[6]

Clinical presentation

The presumptive diagnosis of GORD is made in the presence of typical symptoms, such as heartburn, regurgitation and dysphagia, occurring two or more times a week in patients under the age of 50 years with no other symptoms:[7]

- **Heartburn:** A retrosternal burning sensation or discomfort occurring after meals, when bending over or leaning forward, or when lying in the supine position.
- **Regurgitation:** The spontaneous return of gastric and/or oesophageal contents into the pharynx. Respiratory complications may arise from regurgitation due to regurgitation of gastric content into the tracheobronchial tree.
- **Dysphagia:** One third of patient's experience dysphagia, which refers to a feeling or sensation of food being stuck in the oesophagus, mainly in the retrosternal area.

Other atypical symptoms include coughing, chest pain and wheezing. Complications such as oesophagitis, stricture and Barrett's oesophagus may occur, and these patients should be referred for further diagnostic testing if they do not respond to therapy.[8]

In 50% of cases reflux causes chest pain of a non-cardiac origin and these patients often present to the emergency department thinking that they are having a myocardial infarction. To rule out a cardiac cause, a 24-hour pH testing may be performed, or through the use of oesophageal manometry. A high dosage of a proton-pump inhibitor (PPI) can be used alternatively by utilising a favourable response as an indicator of the non-cardiac origin of the chest pain.[8]

As mentioned in the introduction, GORD may be classified into three distinct categories, namely:

- **Physiological (or functional) gastro-oesophageal reflux:** No underlying factors or conditions are present, with normal growth and development. Pharmacological treatment is generally not necessary, unless lifestyle changes are not successful.
- **Pathological gastro-oesophageal reflux or GORD:** These are patients that regularly experience the above-mentioned symptoms of typical GORD, and they will require proper evaluation and treatment.
- **Secondary gastro-oesophageal reflux:** This is diagnosed when an underlying condition predisposes the patient to gastro-oesophageal reflux.

The management of GORD

GORD is characterised by inflammatory and erosive changes in the normal gut mucosa. The treatment approach to patients with dyspeptic symptoms, as for acid heartburn and GORD, is aimed at:[4,9]

- decreasing the amount of stomach acid that enters the distal oesophagus, usually by neutralising stomach acid, decreasing the production of hydrochloric acid (HCl), increasing the rate at which the stomach empties into the duodenum, and
- relieving the discomfort caused by the 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-HT_4-receptor. These targets may be supported by the simple antacids and the prostaglandin analogues. The pharmacotherapeutic measures may in turn be supported by basic, non-pharmacological intervention strategies.

The treatment of GORD should be individualised, with the goal being to alleviate the symptoms, decrease the frequency of recurrent disease, promote the healing of the mucosal injury and prevent the associated complications.[7]

Non-pharmacological management

A number of suitable dietary recommendations and lifestyle modifications may be proposed but should be individualised for each patient. It is recommendable for patients to refrain from indulging in foods (and drinks) that could trigger the onset of dyspeptic symptoms, such as fats, alcohol, peppermint and spearmint. These foods may decrease the LOSP or increase transient lower oesophageal sphincter relaxation. In contrast to this, spicy foods, orange juice, tomato juice and coffee have a direct irritant effect on the oesophageal mucosa. Smaller meals should also rather be taken more frequently (rather than overindulging at any given point in time) in an attempt to avoid unnecessary gastric distension. Patients should also be advised to avoid the use of nonsteroidal anti-inflammatory drugs (NSAIDs), and other medications with a strong link to the occurrence of dyspepsia (including systemic corticosteroids), wherever possible. If an NSAID or corticosteroid must be used, then the patient should also be given preventative therapy to avoid the uncomfortable dyspeptic symptoms.[7,10-12]

Other non-pharmacological measures may include the following:

- Elevating the head of the bed, which increase oesophageal clearance as well as the pH, may be done with 15–20 cm high blocks underneath the head-side of the bed.
- Weight loss in obese patients (to effectively reduce the associated symptoms).
- Include protein-rich meals in the diet (this augments the LOSP).
- Avoid any significant food intake at least two hours before bedtime, especially in patients that experience any nocturnal symptoms.
- Stop smoking.
- Always take drugs in the upright or sitting position and with a sufficient volume of liquid.

Pharmacological treatment

The pharmacological management (Table I) of GORD should be orientated towards the clinical presentation of the disease as well as the accompanying symptom intensity. The ideal treatment may consist of one or more of the following pharmacotherapeutic options, either alone, sequentially, or in combination:[4,7,9,10,13]

- Simple antacids
- Acid-suppression therapy
- Mucosal or cytoprotective agents
- Pro-motility agents

Simple antacids

Simple antacids, such as those containing aluminium and magnesium, neutralise the hydrochloric acid (HCl) in the stomach and are quite effective as pain relievers. The magnesium-containing antacids cause diarrhoea, while the aluminium-containing agents cause constipation. The combination of magnesium and aluminium will therefore constitute the antacid of choice (e.g. a combination of aluminium hydroxide and magnesium trisilicate). The divalent cations (i.e. Al^{2+} and Mg^{2+}), however, would interact with chelating agents, such as the tetracycline and fluoroquinolone antimicrobials, and several other drug interactions are possible.[4,9]

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 simple antacids. However, care should be taken with these agents, since calcium carbonate may interfere with normal acid-base balance and cause metabolic alkalosis, or it may elicit rebound gastric acid secretion, making it suitable for short-term use only. Meanwhile sodium bicarbonate should be used with caution in patients who require a restricted sodium intake.[4,9]

Dimethicone and simethicone may relieve a 'bloated feeling' by acting as antiflatulent or defoaming agents. They may also be of benefit in the management of intestinal colic in infants and children. However, they do not contribute to the efficacy of the acid neutralisation brought about by the antacids and there is a lack of evidence supporting their chronic use.[4,7,9]

Acid-suppression therapy

Drugs that increase gastric pH fall into two categories, namely the histamine-2 receptor antagonists (H2RAs) and the PPIs, with the latter group constituting the most effective drugs by far.[4,7,9,13]

Histamine 2-receptor antagonists

Blocking the gastric H_2-receptors of parietal cells will reduce stomach acid secretion. These agents are highly selective, inhibitors, capable of suppressing both basal and food-induced acid secretion from these cells, albeit more modestly for the latter and making them less ideal for day-time acid suppression. Ulcer healing rates are significant but not nearly as good as those obtained through the use of the PPIs. In patients with erosive oesophagitis the H2RAs are only effective in fewer than 50% of cases. Cimetidine, ranitidine, famotidine and nizatidine are examples of these selective histaminergic-receptor blockers (the two latter examples are not available locally). Cimetidine has the disadvantage of sometimes producing unwanted antiandrogenic side-effects in male patients (it has a fairly small affinity for androgen receptors). It also has a higher likelihood of multiple drug interactions through its inhibition of cytochrome P450 isozymes. These agents are especially useful in the suppression of nocturnal acid secretion, which largely depends on the physiological actions of histamine.[4,9,13]

Proton-pump inhibitors (PPIs)

These drugs enter the parietal cells of the gastric glands, found in the gastric pits of the stomach lining, where they subsequently and irreversibly inhibit the H^+/K^+-ATPase pump (i.e. the proton pump that is specifically responsible for the H^+-secretion into 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.[4,9,13]

Therefore, these drugs are highly effective in increasing the stomach pH, rapidly relieving the symptoms and achieving good cure rates. They are administered as pro-drugs and are very widely used because of their established, favourable efficacy and safety profiles. PPIs are best taken 30 minutes before breakfast, as a greater quantity of active pumps are available that time of

Table I: Oral acid-lowering agents with indication, cost and dosage recommendations for GORD in adult patients[14]

	As indicated for GORD and/or reflux oesophagitis	Cost per pack*	Cost of the recommended dosage per month (28 or 30 days)
H_2-receptor antagonists (H_2-blockers):			
Cimetidine:			
Adco-Cimetidine®	400 mg QID (120 tablets per month)	400 mg, 60, R51.39	For 120 tablets: R102.78
Bio Cimetidine®	400 mg QID (120 tablets per month)	400 mg, 56, R39.76	For 112 tablets: R79.52
Lenamet®	400 mg QID (120 tablets per month)	400 mg, 56, R39.07	For 112 tablets: R78.14 (A bulk pack size of 500 is also available for R348.84)
Lenamet-OTC®	Only available in the 200 mg-tablet strength for over-the-counter use		
Ranitidine:			
Ranitidine 300 Biotech®	300 mg per day (either in 2 divided dosages, or a single dosage at bedtime); up to 150 mg QID (2 tablets per day, broken in half)	300 mg, 30, R65.71	For 60 tablets: R131.42
Ultak®	300 mg per day (either in 2 divided dosages, or a single dosage at bedtime)	150 mg, 60, R94.77 300 mg, 30, R78.47	For 30 tablets: R78.47
Proton-pump inhibitors (PPIs):			
Omeprazole:			
Adco-Omeprazole®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R157.03	For 28 capsules: R157.03 For 56 capsules: R314.06
Altosec®	20 mg daily (dosage range of 10 to 40 mg daily)	10 mg, 28, R176.41 20 mg, 28, R161.40	For 28 capsules: R176.41 For 56 capsules: R161.40
Corpocid®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R173.85	For 30 tablets: R173.85
Lokit®	20 mg daily (dosage range of 10 to 40 mg daily)	20 mg, 30, R175.32	For 30 capsules: R175.32 For 60 capsules: R350.64
Losec®	20 mg daily (dosage range of 10 to 40 mg daily)	10 mg, 28, R401.91 20 mg, 28, R671.02	For 28 MUPS** tablets: R401.91 For 28 MUPS** tablets: R671.02
Nozer®	20 mg daily (dosage range of 10 to 40 mg daily)	20 mg, 28, R163.04 20 mg, 30, R174.71	For 30 capsules: R174.71
Omez®	20 mg daily (dosage range of 10 to 40 mg daily)	10 mg, 30, R190.85 20 mg, 30, R175.32 40 mg, 14, R155.70 40 mg, 30, R333.69	For 30 capsules: R175.32 For 30 capsules: R333.69
Probitor 20®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R155.66 20 mg, 30, R166.77	For 30 capsules: R166.77
Rapacid®	Only available in the 10 mg-capsule strength for over-the-counter (OTC) relief of heartburn (short-term use)		
Sandoz Omeperazole®	20 mg daily (dosage range of 10 to 40 mg daily)	20 mg, 30, R166.77	For 30 capsules: R166.77 For 60 capsules: R333.54
Lansoprazole:			
Adco-Roznal®	30 mg daily	30 mg, 28, R153.57	For 28 capsules: R153.57
Burnloc®	15 mg daily, as prophylaxis against gastro-oesophageal reflux (or maintenance therapy)	15 mg, 7, R27.46 15 mg, 14, R54.91	For 28 capsules: R109.82
Lancap®	30 mg daily (or 15 mg per day as a maintenance dosage)	15 mg, 14, R55.17 15 mg, 30, R118.22 30 mg, 30, R166.15	For 30 capsules: R166.15
Lansoloc®	30 mg daily (or 15 mg per day as a maintenance dosage)	30 mg, 30, R250.21 (Available as a 15 mg-OTC formulation as well)	For 30 capsules: R250.21
Lansoprazole Unicorn®	30 mg daily (or 15 mg per day as a maintenance dosage)	15 mg, 14, R57.31 15 mg, 30, R122.82 30 mg, 30, R158.88	For 30 capsules: R158.88
Roznal OTC®	15 mg daily, as prophylaxis against gastro-oesophageal reflux (or maintenance therapy)	15 mg, 7, R29.75	For 28 capsules: R119.00
Pantoprazole:			
Conoran®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R126.43 40 mg, 30, R200.61	For 30 tablets: R126.43 For 30 tablets: R200.61
Gastriwin®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R217.98 40 mg, 14, R162.18 40 mg, 28, R324.35	For 28 tablets: R217.98 For 28 tablets: R324.35

Mylan Pantoprazole®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R118.90 40 mg, 30, R185.08	For 30 tablets: R118.90 For 30 tablets: R185.08
Pantocid®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R118.76	For 30 tablets: R118.76 For 60 tablets: R237.52
Pantoloc®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R292.60 40 mg, 14, R275.97 40 mg, 28, R552.05	For 28 tablets: R292.60 For 28 tablets: R552.05
Pantoprazole Unicorn®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 14, R57.59 20 mg, 28, R152.69 40 mg, 28, R229.44	For 28 tablets: R152.69 For 28 tablets: R229.44
Pantor®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R123.25 40 mg, 30, R197.75	For 30 tablets: R123.25 For 30 tablets: R197.75
Pentoz®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 30, R123.28 40 mg, 30, R198.00	For 30 tablets: R123.28 For 30 tablets: R198.00
Pentoz OTC®	Available in a 20 mg tablet (in pack sizes of 7 and 14) delayed-release formulation for the over-the-counter (OTC) relief of heartburn and hyperacidity (short-term use)		
Peploc®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R115.07 20 mg, 30, R123.41 40 mg, 28, R184.81 40 mg, 30, R197.94	For 30 tablets: R123.41 For 30 tablets: R197.94
Peploc OTC®	Available in a 20 mg tablet (in a pack size of 14) tablets for the over-the-counter (OTC) relief of heartburn and hyperacidity (short-term use)		
Prazoloc®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 10, R29.89 20 mg, 30, R89.68 40 mg, 30, R125.09	For 30 tablets: R89.68 For 30 tablets: R125.09
Prazoloc OTC®	AAvailable in a 20 mg tablet (in a pack size of 10) tablets for the over-the-counter (OTC) relief of heartburn and hyperacidity (short-term use)		
Topraflux®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R148.11 40 mg, 28, R220.24	For 28 tablets: R148.11 For 28 tablets: R220.24
Topzole®	20 mg daily (up to 40 mg daily in refractory cases)	20 mg, 28, R219.02 40 mg, 28, R325.59	For 28 tablets: R219.02 For 28 tablets: R325.59
Topzole OTC®	Available in a 20 mg tablet (in a pack size of 7) tablets for the over-the-counter (OTC) relief of heartburn and hyperacidity (short-term use)		
Rabeprazole:			
Pariet®	10–20 mg daily (20 mg daily for erosive oesophagitis)	10 mg, 28, R210.87 20 mg, 28, R421.58	For 28 tablets: R210.87 For 28 tablets: R421.58
Rabemed®	10–20 mg daily (20 mg daily for erosive oesophagitis)	10 mg, 28, R155.63 20 mg, 14, R156.17 20 mg, 28, R312.44	For 28 tablets: R155.63 For 28 tablets: R312.44
Esomeprazole:			
Nectizole®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 30, R210.98 40 mg, 30, R315.03	For 30 'gastro-resistant' tablets: R210.98 For 30 'gastro-resistant' tablets: R315.03
Nesopram®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 30, R238.89 40 mg, 30, R367.47	For 30 'gastro-resistant' tablets: R 238.89 For 30 'gastro-resistant' tablets: R367.47
Nexiam®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 14, R201.28 20 mg, 28, R402.56 40 mg, 14, R310.42 40 mg, 28, R620.84	For 28 MUPS* tablets: R402.56 For 28 MUPS* tablets: R620.84
Nexipraz®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 30, R244.15 40 mg, 30, R376.60	For 30 'gastro-resistant' tablets: R244.15 For 30 'gastro-resistant' tablets: R376.60
Nexmezol®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 14, R113.95 20 mg, 28, R227.91 40 mg, 14, R172.48 40 mg, 28, R345.05	For 28 tablets: R227.91 For 28 tablets: R345.05
Nexomep®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 28, R227.77 40 mg, 28, R345.05	For 28 tablets: R227.77 For 28 tablets: R345.05
Trustan®	20 mg daily (40 mg daily for erosive oesophagitis)	20 mg, 14, R114.16 20 mg, 28, R228.30 40 mg, 14, R174.96 40 mg, 28, R350.00	For 28 'gastro-resistant' tablets: R228.30 For 28 'gastro-resistant' tablets: R350.00

* Prices valid June 2020; ** MUPS – A Multiple-Unit Pellet System for modified drug release

the day. Currently-available examples of PPIs are omeprazole, esomeprazole (the *S*-isomer of omeprazole), lansoprazole, pantoprazole and rabeprazole. The PPIs are still the most effective agents in the management of both nonerosive and erosive GORD, as well as the complications of reflux disease.[4,7,9,13]

Mucosal or 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 perfusion of the gastric mucosa because it is an analogue of prostaglandin E_1 (PGE_1).

Sucralfate forms a protective layer that covers the exposed surface of the ulcer and, in doing so, produces cure rates that are comparable to those obtained with the H_2-receptor antagonists. It should preferably be taken one hour before meals, because it is activated by stomach acid. The viscous paste will cover exposed ulcer or erosive surfaces for up to six hours. Wherever sucralfate is combined with any of the simple antacids, the antacid should be taken half an hour after taking the sucralfate (i.e. on an empty stomach as well).[4,9,13]

Misoprostol is of particular use in preventing the gastrotoxic effects of the NSAIDs. It influences the ratio of acid-to-mucus secretion favourably by increasing gastric mucus secretion while decreasing acid secretion. Care should be taken with this drug, however, since PGE_1 causes uterine contractions, it may be used for termination of pregnancy or the induction of labour, and should therefore be avoided during pregnancy.[4,9,13]

Bismuth compounds may also be used, and may have a variety of beneficial effects, some of which are yet to be fully elucidated. These include the formation of a protective barrier by coating ulcers and erosions in the mucosal lining, stimulating the secretion of mucus, bicarbonate and prostaglandins, as well as its ability to act as an antimicrobial and to bind enterotoxins (hence its usefulness in the management of traveller's diarrhoea and to help eradicate *Helicobacter pylori*).[13]

Pro-motility agents

Metoclopramide acts as an agonist at 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-HT4-receptor agonist, which is unrelated to the abovementioned two drugs. It has the disadvantage of causing potentially serious cardiac side-effects, such as ventricular dysrhythmias (by causing QTc-interval prolongation), especially when its own metabolism is inhibited (through various drug interactions, for instance). Access to this drug has been restricted and it should be used with extreme caution.[4,9,13]

Bethanechol is a parasympathomimetic drug, which selectively stimulates muscarinic receptors (of the M_3-subtype). In the gastrointestinal tract (GIT) this causes smooth muscle contraction but produces relaxation of the sphincters. Bethanechol therefore stimulates the functional contraction of the GIT (i.e. it increases intestinal motility). A different approach with a similar outcome on the motility of the GIT would be to use neostigmine. Erythromycin also has pro-kinetic properties. It acts as a direct stimulator of the motilin receptors.[4,13]

The usefulness of these agents in GORD is limited, with metoclopramide and domperidone being reserved for patients with regurgitation and refractory heartburn.[13]

Table I provides an overview of the different oral, acid-lowering agents on the local market (with a specific indication and dosage recommendation for reflux oesophagitis, as part of GORD.)

Conclusion

Physicians and other healthcare professionals should be aware of GORD and its treatment strategies, because it constitutes a significant disease burden worldwide. In the management of GORD various agents and classes of agents are available, either for the management of the associated symptoms, or for the treatment thereof. It has been shown that the PPIs are more effective than the H2RAs in managing GORD and are also superior to placebo in patients with GORD-related symptoms. Specific drug selection within the PPI group, should be based on individual adverse effect profiles and the expected onset of action.

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