Hiatus hernia and gastro-oesophageal reflux disease

Rishi K Fofaria

Danielle L Morris

Abstract

Gastro-oesophageal reflux disease (GORD) is a common condition with a prevalence of 10—25% in Europe and a rising prevalence in developing nations. GORD is typified by the symptomatic passage of gastric contents into the oesophagus with or without mucosal injury. Hiatus hernias usually contribute to the pathophysiology of GORD and the presence of an 'acid pocket' can worsen symptom severity. Rather than a spectrum of disease progression, GORD is best considered as a group of related syndromes: endoscopy-negative disease, oesophagitis (with or without complications, such as strictures), Barrett's oesophagus and oesophageal adenocarcinoma. While a 'classical' presentation with retrosternal discomfort, volume reflux or mild odynophagia is common, it is important to recognize extra-oesophageal manifestations of the disease, especially respiratory and laryngeal symptoms. The mainstay of management is symptom control through lifestyle modification and acid suppression therapy. However, in selected patients, anti-reflux surgery may be indicated, and novel medical and surgical treatments are being actively researched.

Keywords Anti-reflux surgery; dyspepsia; gastro-oesophageal reflux disease; hiatus hernia; oesophagitis; proton pump inhibitors

Definitions and epidemiology

Gastro-oesophageal reflux disease (GORD) is a condition that develops from the retrograde reflux of acidic (or bilious) gastroduodenal contents into the oesophagus, or beyond, causing troublesome symptoms and/or complications.[1,2] However, symptoms may be transient and complications such as oesophagitis may be asymptomatic. The prevalence of GORD is rising globally; it is between 10 and 25% in Europe and even higher in the Middle East, but consistently less than 10% in East Asia.[3] GORD can affect all ages, but is more common in those aged over 40 years. Although GORD itself has no gender predilection, oesophagitis (2:1) and Barrett's oesophagus (10:1) are more common in males.

The 'sliding' type of hiatus hernia, in which the mucosal gastro-oesophageal junction (GOJ) slides superiorly to rest wholly above the diaphragm, has an estimated prevalence of 25—50%, rising with age and obesity, and its incidence is strongly correlated to the concurrent diagnosis of GORD.[4] Almost all hiatus hernias (95%) are of this type. Other types of hiatus hernia — the 'para-oesophageal' and 'rolling' types — are non-contributory to GORD but often require surgical correction. It is important to remember that one can have a hiatus hernia with no symptoms of GORD and vice versa.

Rishi K Fofaria *BSc MRCP is a Specialist Registrar in Gastroenterology in London. He trained at University College Medical School and Imperial College, London, UK. Current interests: Barrett's oesophagus and metabolic disease. Competing interests: none declared.*

Danielle L Morris *MSc MD FRCP is a Consultant Gastroenterologist at East and North Herts NHS Trust and University College Hospital, London. She trained at University College Medical School and the Middlesex Hospital, London, UK. Current interests: Barrett's oesophagus and oesophago-gastric cancer. Competing interests: none declared.*

Risk factors

Risk factors for GORD include genetic predisposition (up to 30% of the risk),[5] abdominal obesity, pregnancy, hiatus hernia, delayed gastric emptying and age. Less common factors include medications, such as calcium channel blockers, anticholinergics and nitrates, which reduce lower oesophageal sphincter (LOS) pressure. Cigarette smoking, and consumption of alcohol and certain foods (e.g. mints and citrus fruits) and drinks (e.g. coffee and carbonated drinks) are thought to contribute but the association is not proven.

Pathophysiology

The pathophysiology of GORD is multifactorial and relates to an imbalance between injurious factors sensitizing the mucosa (caustic gastric and duodenal contents) and defensive factors preserving the competency of the GOJ and promoting acid clearance. The anti-reflux barrier comprises the LOS (more accurately the distal oesophageal 'high pressure zone'), which passes through the hiatus created by the right diaphragmatic crus making up the external sphincter.

Mechanism of reflux

Physiological reflux from LOS relaxation during swallowing is immediately neutralized by alkaline bicarbonate-rich saliva and cleared by peristalsis.[6] At other times, reflux occurs spontaneously and transiently after vagally (GABA) mediated stretch responses.[7] Post-prandially, gastric distension allows natural gas venting to occur as a result of increased transient lower oesophageal sphincter relaxations (TLOSR).[8] In GORD, a greater proportion of these sphincter relaxations result in reflux of acid.[9] Furthermore, certain foods, including chocolate, alcohol and peppermint, and colonic fermentation of carbohydrates have also been observed to increase the rate of TLOSR through release of glucagon-like peptide-1.[10] Obesity, hiatus hernias and even right-sided recumbence have all been associated with higher TLOSR (Figure 1).

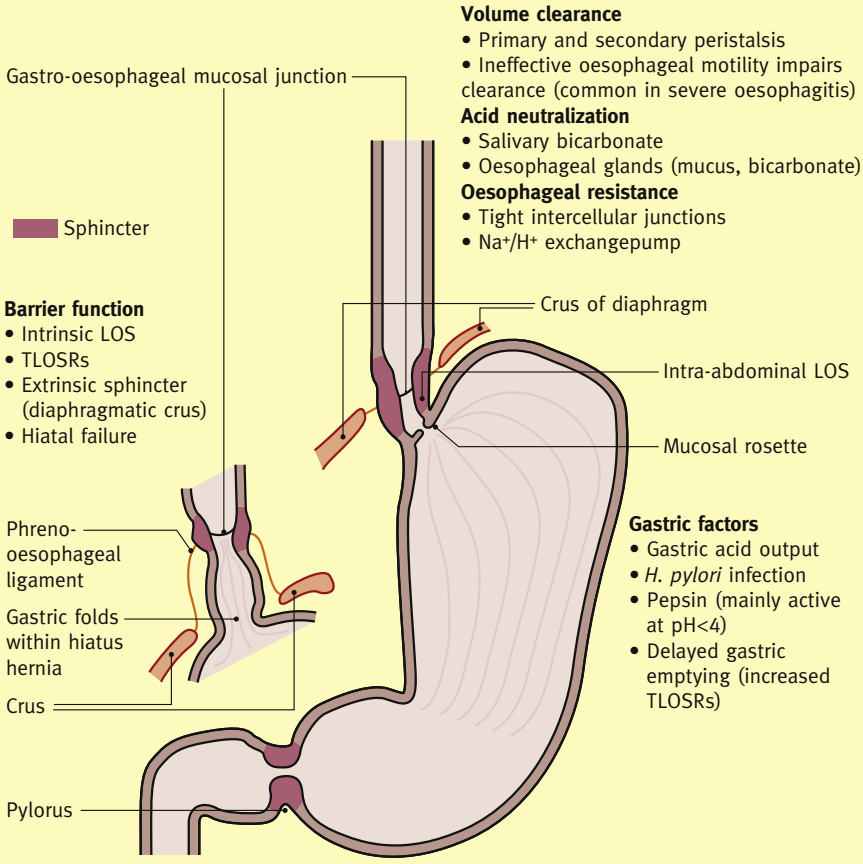
Other mechanisms accounting for reflux events include disordered peristalsis, acutely raised intra-abdominal pressure and an atonic LOS allowing the free passage of acid.[6]

The above mechanisms are exacerbated by hiatus hernias and explained by the 'acid pocket' theory. Whereas gastric contents are at their least acidic following food ingestion, the proximal stomach and hiatal 'pocket' often escapes this buffering mechanism, allowing a very acidic refluxate to be entrapped above the diaphragm and even to cross the squamo-columnar junction, resulting in acidic reflux during TLOSR.[11]

Other pathophysiological factors

Delayed gastric emptying and the reflux of bile acids from the duodenum can act synergistically with gastric acid to worsen

A summary of factors involved in gastro-oesophageal reflux disease



LOS, lower oesophageal sphincter; TLOSR, transient lower oesophageal sphincter relaxation.

Reproduced from Cram M, de Caestecker J. Hiatus hernia and gastro-oesophageal reflux disease. *Medicine 2011; **39**(3); 132-136)*

Figure 1

GORD. The effect of *Helicobacter pylori* in changing the pH of gastric refluxate may even protect against GORD, but studies have not confirmed an increase in occurrence after eradication.[12]

Importantly, symptoms can occur despite a less acidic refluxate. This is termed non-acid reflux and is often attributed to high proximal regurgitation and a hypersensitized oesophagus with abnormal oesophageal mechanoreceptor responses.[13]

Disease course and natural history

It is postulated that GORD should no longer be approached as a continual spectrum of disease correlating to severity of mucosal injury, in which endoscopy-negative reflux disease is classed as mild, oesophagitis as moderate and Barrett's oesophagus as severe.[14] This is because sequential progression through these stages is rarely seen, with only up to 30% developing oesophagitis and only 1–13% developing Barrett's oesophagus.[15] Research shows that oesophageal physiology and mucosal biology is often not shared and that the response to therapy and risk of malignancy does not change continuously.[16]

Uncomplicated GORD does have a chronic and relapsing course with up to two-thirds of patients taking medications continuously or intermittently up to 10 years after diagnosis. In those who stopped taking medications, only 20% had resolution of symptoms.[17] Mortality is extremely low at 0.1/100,000 persons/year.

Diagnosis and differential diagnosis

GORD (and indirectly hiatus hernia) is often diagnosed from the history alone with or without the presence of risk factors as outlined above.

Classical symptoms: heartburn (particularly to typical foods), regurgitation, volume reflux, mild odynophagia or dysphagia.

Atypical and extra-oesophageal symptoms: coughing, wheezing, hoarseness (Cherry–Donner syndrome), non-cardiac