GASTRO-OESOPHAGEAL REFLUX DISEASE Aetiology

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GORD is defined by the ‘Montreal definition’ as a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications. The aetiology of GORD can be explained by the interaction between the reflux barrier and the pressure di ff erence between the thoracic and abdominal cavity . The reflux barrier consists of the crural diaphragm and the LOS. Physiological relaxation of the LOS in response to stretching of the gastric fundus, particularly after a meal to allow venting of swallowed air, is termed transient LOS relaxations (TLOSRs). An increased number of TLOSRs and a more compliant LOS would increase reflux. Delay in acid refluxate clearance from the oesophagus, as a result of defective oesophageal motility , also contributes to oesophageal exposure. Hiatus hernia is associated with GORD; it is formed when the weakened phreno-oesophageal ligament and widened cru ral opening allow the proximal stomach to herniate through the diaphragmatic hiatus. Ageing, connective tissue disease and elev ated intra-abdominal pressure (e.g. central obesity , ments, chronic straining) will further pregnancy , tight gar aggravate the hernia. An acid pocket is an area of unbu ff ered gastric acid that accumulates in the proximal stomach after meals and serves as a reservoir for acid reflux. Together with a hiatus hernia, it can exacerbate the severity and symptoms of GORD. The overall global incidence of GORD is increasing and has a predominant regional distribution in the Americas and ope; up to 33% of the population is a ff ected, compared with Eur - <10% in Asia. The increase in GORD incidence is attributed to the global increase in obesity and declining rates of Helico - bacter pylori infection. This increasing prevalence of GORD coincides with the increased mortality rates from oesophageal adenocarcinoma. Central obesity , independent of body mass index, is a risk factor for developing Barrett’s oesophagus and adenocarcinoma, while H. pylori may have a preventive role.

(a) Figure 66.10 Endoluminal functional lumen imaging planimetry reading before with achalasia. The column of numbers on the right-hand side of each panel shows the diameter of each 0.5-cm segment along the narrowest segment is indicated by the green circle). This is used to calculate the cross-sectional area. The distensibility index (DI) (red circle) is 3 calculated by dividing the cross-sectional area (mm) by the intra-balloon pressure (mmHg) (blue circle). The DI improves from 0.8 mm 3 to 4.2 mm /mmHg after POEM, indicating that the lower oesophageal sphincter has become more ‘compliant’. (a) Figure 66.11 (a) Wireless capsule used to measure pH data in pH monitoring, with the capsule in the delivery catheter before deployment. (b) Catheter used to measure pH as well as impedance. The catheter is placed through the nostril. The most distal sensor is placed 5 cm above the upper border of the lower oesophageal sphincter measured on high-resolution manometry. (b) (a) and after (b) peroral endoscopic myotomy (POEM) in a patient catheter (the 3 /mmHg (b)

8.0 A pH1 12:00AM 12:00PM 12:00AM 0.0 Interpretation / Findings DeMeester Score Acid exposure time 2 h 42 min 40.2 Day1 off PPI 1 h 17 min 16.9 Day2 off PPI 15 min 4.3 Day3 on PPI 0.3 Day4 on PPI Overall • DeMeester Score = 15.9 Figure 66.12 A wireless pH monitoring trace over 4 days. A re /f_l ux episode is de /f_i ned when the pH drops to less than pH 4 (below the horizontal blue line). This patient had stopped taking a proton pump inhibitor (PPI) 2 weeks before the study. He was then given a daily dose of PPI starting on day 3 of the examination. The DeMeester scores were abnormal (>14.7) on days 1 and 2 but returned to the normal range after acid suppression by the PPI. The symptom association probability (SAP) was 100% on days 1 and 2, meaning that every time there was acid re /f_l ux the patient experienced symptoms. SI, symptom index; SSI, symptom sensitivity index. LOSd, lower oesophageal sphincter, distal; LOSp, lower oesophageal sphincter, proximal; PIP , pressure inversion point; UOS, upper oesophageal sphincter. Auto Range 11:06:20.915 k /uni03A9 7.00 Z1 2.05 0.00 k /uni03A9 7.00 Z2 1.50 0.00 k /uni03A9 7.00 Z3 5.90 0.00 7.00 k /uni03A9 1.73 Z4 0.00 A 7.00 k /uni03A9 Z5 0.53 0.00 7.00 k /uni03A9 Z6 0.41 0.00 A 9.0 pH 119 pH1 3.4 11:06:20 11:06:30 0.0 Display Mode Play 15:00 18:00 21:00 00:00 03:00 06:00 09:00 12:00 Figure 66.13 pH impedance data tracing. A drop in impedance signi /f_i es liquid within the oesophageal lumen while a belch (with air) results in a rise in impedance. In this /f_i gure, the x-axis represents time. There is a sequential drop of impedance from the distal sensor (Z6) to the more proximal sensor (Z3) (indicated by the red arrow and the shaded boxes), signifying a liquid re /f_l ux episode. At the same time, the pH sensor detected pH < 4 (lowest row), indicating that the re /f_l ux episode is an acid re /f_l ux. LOSd, lower oesophageal sphincter, distal; LOSp, lower oesophageal sphincter, proximal; PIP , pressure inversion point; UOS, upper oesophageal sphincter. UOS 19.0 Oesophagus 36.1 LOSp 41.1 PIP 42.2 Diaphragm LOSd 44.8 Stomach 12:00PM 12:00AM 12:00PM 12:00AM 12:00PM No. of total reflux/post-prandial/supine SI, SSI, SAP 77.8, 12.3, 100.0 57/46/1 85.7, 11.1, 100.0 54/48/0 0 10/9/0 0 0 0 Pharynx 53 UOS 20.0 26.2 28.2 30.2 Oesophagus 34.2 36.2 38.2 39.2 40.2 42.2 LOSp 44.2 PIP 45.6 LOSd 47.1 120 Stomach 11:06:40 11:06:50 11:07 pH = 0.0 pH 9.0 Z = 0.00 k /uni03A9 7.00 Meal Supine Mode Anatomy Range Med Symptom Anatomy Source:

as a result of H. pylori -related corpus gastritis or gastric atrophy would lead to decreased oesophageal acid exposure.

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