2
Esophageal Disorders

2.1 Diverticula

The following diverticula can be found in the esophagus

- Zenker's diverticula,
- Pulsion diverticula
- Traction diverticula
- Embryonic malformations (congenital diverticula) – extremely rare
- Pseudodiverticulosis in chronic inflammation: these diverticula are strictly intramural and correspond to enlarged submucosal esophagal glands

2.1.1 Pulsion Diverticula

They are formed by mismatch of intraluminal pressure and esophageal wall. Accordingly, they typically occur above sphincters and mostly consist of pseudodiverticula without muscle layer. Within the distal 10 cm of the esophagus, they are called epiphrenic diverticula and are associated with motility disorders. Above the upper esophageal sphincter, the wall of the hypopharynx can expand, which results in Zenker's diverticula.

2.1.2 Zenker's Diverticula

The most common diverticulum can be suspected with a history of dysphagia, regurgitation of undigested food, chronic cough/aspiration and fetor ex ore. Typically, Zenker's diverticula become symptomatic at the age of 70–80 years and can lead to weight loss and malnutrition.

Cervical borborygmi are pathognomonic. Diagnosis is made by barium meal examination. Due to the anatomy, endoscopy leads axially always directly into the thin diverticular pouch. Blind or side-viewing endoscopies (EUS, ERCP) are, therefore, dangerous.

Indication of therapy is based on symptoms. Three competing treatment modalities exist: open surgical resection, endoluminal stapler-assisted therapy or the flexible endoscopic version with transection of the stem between the diverticulum and the esophagus. All 3 methods have a success rate of >80%; however, flexible endoscopic

treatment can be carried out under sedation and with a relatively short intervention time with the lowest morbidity.

2.1.3 Traction Diverticula

Pulling of the wall from the outside, usually by mediastinal scarring or inflammation, gives rise to these real diverticula, which are mostly located in the midesophageal region. From an etiological perspective, these occur along with TB, histoplasmosis, lymphomas and postoperatively.

2.2 Inflammation of the Esophagus (Including Eosinophilic Esophagitis)

2.2.1 Gastroesophageal Reflux Disease

GERD is defined as a rise in gastric juice into the esophagus causing symptoms at least 2 days per week.

- Two forms of GERD are distinguished:
  - Endoscopically or histologically detectable epithelial defects: "erosive reflux disease" (ERD)
  - No evidence of endoscopic defects: non-ERD
- In the Western industrialized countries, 14–20% of the population suffers from GERD.
- In approximately 60% of them, no erosions can be detected endoscopically (non-ERD).
- In the remaining 40% with ERD, a Barrett's esophagus is found in 5%.
- 10% of them develop adenocarcinoma (Barrett's carcinoma).

Gastroesophageal reflux is physiological to a certain degree. This acidic reflux is normally transported back into the stomach by secondary contractions of the esophagus, which fulfills a cleaning function between the swallowing of food boluses.

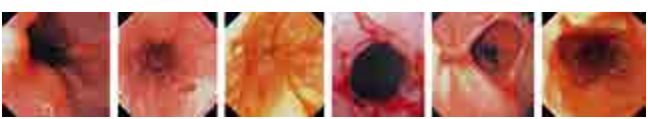
In case of GERD, this mechanism is usually not sufficient: The esophageal epithelium remains in contact with the gastric acid for a longer period of time.

Other mechanisms play also a role

- Due to reduced pressure and inappropriate relaxation of the lower esophageal sphincter, gastric contents enter the esophagus in larger amounts or for a longer time period.
- An axial hiatus hernia interferes with the function of the sphincter.
- An increase in abdominal pressure, for example due to overweight or pregnancy (in particular in the third trimester), promotes gastroesophageal reflux and increases its amount.
- Certain foods/diets may trigger acid reflux.
- Drugs (calcium channel blockers, nitroglycerin preparations, theophylline) may cause acid reflux.
- In rarer cases, delayed gastric emptying, for example in diabetic neuropathy, increases reflux.

2.2.2 Classification of Reflux Esophagitis according to Savary and Miller

Figure 2.1 depicts the different grades of reflux esophagitis. Esophageal and extraesophageal complaints are listed in Table 2.1.



Grade 0 No macroscopic mucosal changes

Grade I Isolated mucosal lesions

Grade II Longitudinally confluent lesions

Grade III Lesions over the entire circumference

Grade IV Complication stage: ulceration with scarring (fibrosis, stenosis)

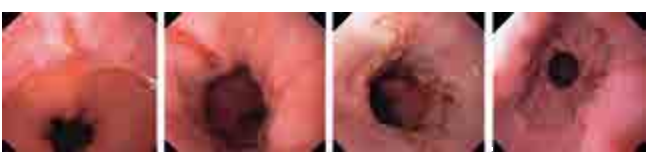
Grade V Barrett's esophagus

Fig. 2.1. Reflux esophagitis: Savary-Miller [1].

Table 2.1. Frequency of esophageal/extraesophageal complaints

Esophageal complaints	
Heartburn (when lying and after meals)	60%
Sensation of pressure/burning behind the sternum (differential diagnosis: coronary heart disease)	57%
Regurgitation of acid and partially digested food	55%
Pain/burning sensation below the xiphoid	55%
Belching	45%
Nausea	35%
Odynophagia	10%
Extraesophageal complaints	
Tooth enamel erosion	35%
Laryngitis and hoarseness	25%
Chronic cough	20%
Intensification of bronchial asthma	10%
Sinusitis, recurrent otitis media	Very rare

The Los Angeles classification distinguishes only the extent of the erosions (Fig. 2.2). Strictures or ulcers play no role here. Stages A and B of this classification correlate with grade I according to Savary and Miller, stage C with grade II and stage D with grade III.



Stage A
Erosion(s) ≤5 mm
One mucosal fold

Stage B
Erosion(s) >5 mm
One mucosal fold

Stage C
Erosions over several mucosal folds
≤75% of the circumference

Stage D
Erosions over several mucosal folds
>75% of the circumference

Fig. 2.2. Reflux esophagitis: Los Angeles classification [2].

The Montreal classification distinguishes between esophageal and extraesophageal syndromes (Fig. 2.3). In GERD patients without alarm systems, gastroscopy is not always the first choice of therapy (Fig. 2.4, 2.5).

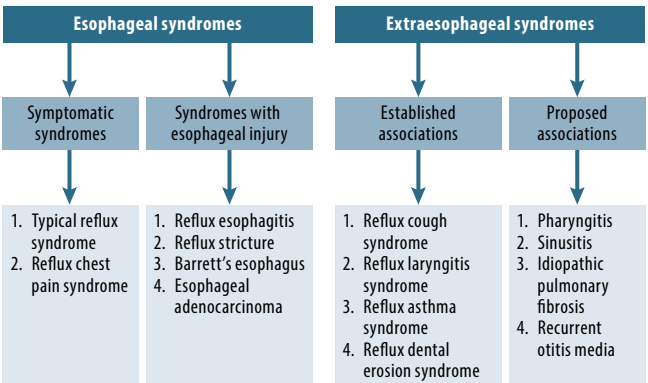


Fig. 2.3. Montreal classification.

Gastroscopy revealed clinically significant findings in 38% of patients with reflux symptoms without alarm symptoms (Fig. 2.4) in a study by Peng et al. [3], whereas the the German Society for Digestive and Metabolic Diseases (DGVS) recommended PPI treatment (Table 2.2) for patients without alarm symptoms before gastroscopy in 2005 (Fig. 2.2) [4].

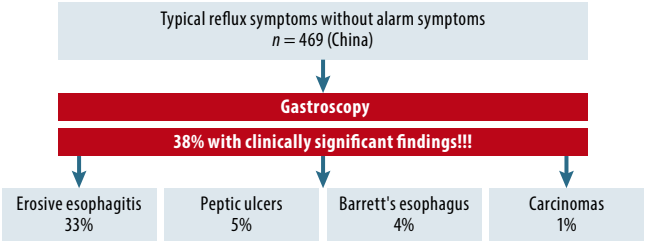


Fig. 2.4. Gastroscopy despite missing alarm symptoms [3].

Table 2.2. Common drugs for relief of reflux symptoms

Drugs	Starting dose/day	Maintenance dose/day
Proton pump inhibitors		
Esomeprazole	40 mg	20 mg
Lansoprazole	30 mg	15 mg
Omeprazole	40 mg	20 mg
Rabeprazole	20 mg	10 mg
Pantoprazole	40 mg	20 mg
H_2 receptor antagonists		
Ranitidine	300 mg	150 mg
Famotidine	40 mg	20 mg
Nizatidine	300 mg	150 mg
Roxatidine	150 mg	75 mg
Antacids		
Sucralfate	4 × 1 g	2 × 1 g
Magnesium hydroxide	Often combinations of the individual active ingredients in finished formulations; intake several times a day	
Magnesium trisilicate		
Aluminum hydroxide		

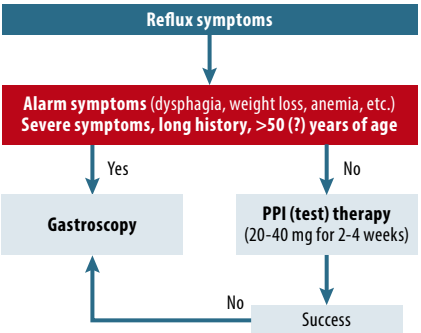


Fig. 2.5. Approach to diagnostic clarification [4].

2.2.3 Eosinophilic Esophagitis

Diagnosis

- Case history
- Food allergies
- Atopy
- Gastroscopy with distal and proximal biopsies (4 each); pliers with spike and under suction; >15 eosinophils/high-power field

Therapy

- Topical steroids: 1 mg fluticasone (1-0-1) p.o. for 2 weeks, then 250 µg (1-0-1) p.o. for 1 year, then fasted for at least 30 min
- Optionally PPIs (PPI responder subgroup)
- Bouginage
- Systemic steroids with caution
- Immunomodulators only in individual cases and in studies
- Consultations/studies: inquiries at the motility group of the University Hospital Zurich (USZ)