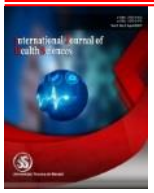

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Acid Reflux and ENT Manifestations: A Review of Laryngopharyngeal Reflux Disease

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Abstract

Laryngopharyngeal reflux disease (LPRD), an extraesophageal manifestation of gastroesophageal reflux disease (GERD), occurs when gastric contents reach the larynx and pharynx, frequently without classic heartburn or regurgitation ("silent reflux"). This condition accounts for up to 10% of otolaryngology consultations and affects 15–30% of patients with laryngeal complaints. The laryngopharyngeal mucosa is highly susceptible to injury from acid, pepsin, bile acids, and trypsin, leading to direct mucosal damage and vagally mediated reflexes. Common ENT manifestations include hoarseness, chronic cough, throat clearing, globus pharyngeus, excessive mucus, and sore throat, with less frequent associations such as chronic rhinosinusitis, subglottic stenosis, and laryngeal granulomas. The diagnosis incorporates clinical history, the Reflux Symptom Index (RSI >13), the Reflux Finding Score (RFS >7) on laryngoscopy, and objective testing using 24-hour hypopharyngeal–esophageal multichannel intraluminal impedance-pH monitoring as the reference standard. The multimodal, step-by-step management process begins with dietary and lifestyle modifications and progresses to proton-pump inhibitors, alginates, and, in refractory cases, anti-reflux surgery or adjunctive therapies. Recent international consensus statements emphasize the importance of objective confirmation of reflux to reduce overtreatment. This review compiles the latest information on pathophysiology, diagnosis, and treatment to enhance patient outcomes. It emphasizes the necessity of interdisciplinary cooperation and puts more effort into non-acid reflux components and novel biomarkers.

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Contents

1 Introduction

Acid reflux, primarily as gastroesophageal reflux disease (GERD), involves retrograde flow of gastric contents into the oesophagus, classically causing heartburn and regurgitation. A substantial proportion of patients, however, experience extraoesophageal manifestations, particularly in the ear, nose, and throat (ENT) region, collectively termed laryngopharyngeal reflux (LPR) or laryngopharyngeal reflux disease (LPRD). In LPRD, refluxate reaches the laryngopharynx and upper aerodigestive tract, often without typical GERD symptoms ("silent reflux"). LPRD accounts for up to 10% of otolaryngology consultations and is implicated in 15–30% of patients presenting with laryngeal complaints (Cui et al., 2024; Brown et al., 2025).

Although LPRD is often thought of as an extraoesophageal manifestation of GERD, new research indicates that it should be considered a separate entity due to differences in pathophysiology, symptom profile, and response to treatment. Overlap is frequent: up to 46% of GERD patients have LPR characteristics, and 46–53% of LPRD patients have concurrent GERD. Due to persistent symptoms that affect swallowing, voice, and quality of life, the condition has a substantial socioeconomic burden (Chen et al., 2023; Krause & Yadlapati, 2024).

2 Pathophysiology

The laryngopharyngeal mucosa is exquisitely sensitive to injury compared with esophageal mucosa. The oesophagus tolerates up to 50 reflux episodes daily, whereas the larynx may sustain damage from as few as 3–4 episodes. Two primary mechanisms underlie LPRD: direct mucosal injury by refluxate and vagally mediated reflexes (Cui et al., 2024).

3 Direct Injury

Results from exposure of the delicate ciliated epithelium to gastric acid, pepsin, bile acids, and trypsin. Unlike the oesophagus, the larynx lacks robust protective mechanisms such as bicarbonate secretion or thick mucus. At pH levels as high as 8, pepsin maintains its proteolytic activity, denaturing proteins and sustaining inflammation. Bile acids cause DNA damage, cytokine release, and the epithelial–mesenchymal transition, especially when they are protonated in acidic environments. Additionally, many LPR patients show decreased expression of salivary epidermal growth factor and carbonic anhydrase III, which hinders mucosal repair and acid neutralization (Cui et al., 2024).

4 Reflex Mechanisms

involve the stimulation of esophageal vagal afferents by acid, which causes laryngeal reactions such as bronchospasm, coughing, and throat clearing. These actions create a vicious cycle by making mucosal edema worse. Proximal migration of refluxate is facilitated by transient relaxations of the lower and upper esophageal sphincters, reduced esophageal clearance, and sphincter dysfunction (Lien et al., 2023).

Clinical ENT Manifestations
LPRD symptoms are nonspecific and overlap extensively with allergies, post-nasal drip, vocal misuse, and infection. Common presentations include:

- Hoarseness or dysphonia
- Chronic cough or throat clearing
- Globus pharyngeus (lump-in-throat sensation)
- Excessive throat mucus or post-nasal drip sensation
- Sore throat or pharyngeal irritation

5 Dysphagia or Halitosis

Less frequent but recognized associations include chronic rhinosinusitis, otitis media with effusion, Eustachian tube dysfunction, subglottic stenosis, laryngeal granulomas, and an increased risk of laryngeal carcinoma. LPR may also contribute to refractory asthma and chronic cough (10–40% of cases in selected series) (Chen et al., 2023; Brown et al., 2025).

Laryngoscopic findings, often quantified by the Reflux Finding Score (RFS), include posterior commissure hypertrophy, interarytenoid edema/hyperaemia, vocal-fold edema, ventricular obliteration, and diffuse laryngeal erythema. These signs are sensitive but lack specificity and may occur in asymptomatic individuals (Krause & Yadlapati, 2024).

6 Diagnosis

No single test is definitive for LPRD; diagnosis integrates clinical history, laryngoscopy, and objective testing. The Reflux Symptom Index (RSI), a validated nine-item patient-reported outcome measure, scores symptoms from 0–45; a score >13 is suggestive of LPR. When combined with an RFS >7 on laryngoscopy, the probability of LPR increases, although these tools should not be used in isolation owing to modest specificity (Belafsky et al., 2002; Belafsky et al., 2001).

The reference standard remains 24-hour hypopharyngeal–esophageal multichannel intraluminal impedance-pH monitoring (HEMII-pH), which detects both acid and non-acid reflux events reaching the pharynx. Emerging non-invasive biomarkers such as salivary pepsin testing show promise but require further validation. Due to high placebo response rates and frequent non-response (up to 40%), empirical proton-pump inhibitor (PPI) trials are not recommended as first-line diagnostics. Given the complex nature of laryngeal symptoms, a multidisciplinary evaluation involving otolaryngology, gastroenterology, and allergy specialists is advised (Lien et al., 2023; Treat & Vaezi, 2026).

In patients with isolated laryngeal symptoms in particular, recent consensus statements stress the importance of objective confirmation of reflux before long-term therapy (Cuff & Yadlapati, 2025; Yadlapati et al., 2026).

7 Management

The cornerstone of the multimodal, step-by-step treatment is dietary and lifestyle changes. Lifestyle and dietary interventions — Head-of-bed elevation, avoidance of meals 2–3 hours before recumbency, weight loss, elimination of triggers (caffeine, alcohol, spicy/fatty foods, chocolate, mint), loose clothing, and gum chewing

to increase salivary flow are first-line measures that significantly improve RSI scores when adhered to (Lechien et al., 2021).

Pharmacologic therapy — Twice-daily PPIs for 8–12 weeks remain the mainstay for acid suppression. However, non-acid reflux (pepsin, bile) explains many treatment failures. Adjunctive alginates provide a physical barrier and are effective against both acid and non-acid episodes. Other agents include H_2-receptor antagonists, baclofen (to reduce transient sphincter relaxations), and mucosal protectants. Emerging therapies target pepsin or protease-activated receptor-2 pathways (Lin & Peng, 2026; Treat & Vaezi, 2026).

Surgical and adjunctive options — For PPI-refractory, objectively confirmed LPR, anti-reflux surgery (fundoplication) offers durable symptom control but carries risks of dysphagia and gas-bloat syndrome. Non-surgical devices (external upper-esophageal-sphincter compression), speech-language pathology for behavioral modification, and psychological support for throat-clearing habits provide additional benefit (Krause & Yadlapati, 2024). Monitoring for possible side effects is necessary when using PPIs for an extended period (e.g., bone density loss, infections). Prognosis is favourable with adherence, although symptom recurrence is common without sustained lifestyle changes (Treat & Vaezi, 2026).

8 Conclusion

Acid reflux frequently manifests in the ENT domain as LPRD, producing chronic, nonspecific symptoms that may occur independently of classic GERD. Heightened clinician awareness, coupled with objective testing (rather than symptom- or laryngoscopy-based diagnosis alone), is essential to improve outcomes and avoid overtreatment. Ongoing research into non-acid reflux components, novel biomarkers, and personalized therapies, informed by recent international consensus statements, promises refined management of this prevalent yet challenging condition. Multidisciplinary collaboration between otolaryngologists and gastroenterologists remains critical for optimal patient care.

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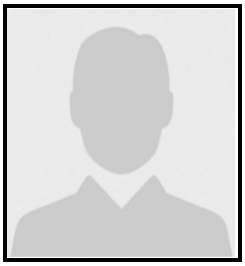

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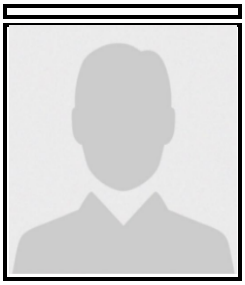

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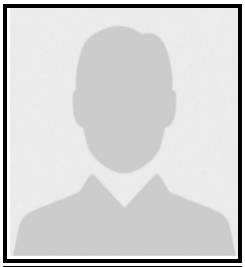

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