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**Editorial**

# Laryngopharyngeal reflux disease (LPRD), a silent reflux - An overview

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## 1. Introduction

Laryngopharyngeal reflux disease (LPRD) is a disorder characterized by the backflow of stomach acid into the laryngopharynx, resulting in inflammation and damage to the mucosal lining. Unlike gastroesophageal reflux disease (GERD), which mainly affects the esophagus, LPRD predominantly impacts the larynx, pharynx, and airways, causing symptoms like persistent hoarseness, chronic coughing, a lump-like sensation in the throat (globus pharyngeus), and frequent throat clearing. Affecting 4–30% of patients in otolaryngology clinics, LPRD is a major contributor to voice-related issues, though its diagnosis is often delayed due to vague symptoms and similarities with other conditions. This review consolidates the latest research on LPRD, discussing its underlying mechanisms, symptom profiles, diagnostic methods, and management options, while incorporating insights from recent studies.

## 2. Definitions

Laryngopharyngeal Reflux (LPR): LPR occurs when stomach acid flows back into the larynx and pharynx. Laryngopharyngeal Reflux Disease (LPRD) refers to the symptoms, physical signs, and tissue damage in the throat, voice box, and related respiratory structures caused by this acid exposure. LPR is frequently mistaken for gastroesophageal reflux (GER).

Gastroesophageal Reflux (GER): GER involves the backward movement of gastric contents into the esophagus. When this reflux becomes excessive and leads to tissue injury or noticeable symptoms, it is classified as gastroesophageal reflux disease (GERD).

Extraesophageal Reflux: Also known as extraesophageal reflux disease (EERD), this condition involves GERD symptoms that manifest beyond the esophagus and stomach. These symptoms can affect the laryngopharynx, airways, nasal passages, sinuses, and even the middle ear.

## 3. Pathophysiology and Mechanisms

The main defense against gastroesophageal reflux (GER) is the lower esophageal sphincter (LES), situated at the diaphragmatic hiatus, which serves as the primary barrier against stomach acid backflow. Additional structures that contribute to reflux prevention include the intra-abdominal portion of the esophagus, the angle of His (gastroesophageal angle), the diaphragmatic crura, and the phrenoesophageal ligament. Impaired gastric motility, particularly delayed emptying, can also increase the risk of GERD.[1] Therefore, the mechanisms protecting against reflux involve both anatomical and physiological factors:

## 4. Anatomical Factors:

1. Lower esophageal sphincter (LES).
2. Intra-abdominal segment of the esophagus.
3. Angle of His (gastroesophageal angle).
4. Mucosal folds in the distal esophagus.
5. Phrenoesophageal ligament.
6. Constrictive action of the diaphragmatic crura.

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**5. Physiological Factors**

1. Efficient esophageal peristalsis to clear refluxed material.
2. Rapid gastric emptyin

**6. Direct Mucosal Injury**

LPRD results from the reflux of gastric acid, pepsin, bile acids, and trypsin into the laryngopharynx, where the mucosa lacks protective mechanisms present in the esophagus. Pepsin remains active at pH ≤ 6.5, causing cellular damage even in weakly acidic environments. Bile acids exacerbate injury by disrupting epithelial barriers, while trypsin activates inflammatory pathways.

**7. Reflex-Mediated Effects**

Vagal stimulation by esophageal acid exposure triggers chronic cough and throat clearing, perpetuating mucosal trauma. Indirect injury via chemoreceptor activation leads to laryngeal hypersecretion and globus sensation.

**8. Non-Acid Reflux**

Up to 50% of LPRD cases involve weakly acidic or gaseous reflux, complicating diagnosis and treatment. It has been found that non-acid reflux is associated with symptoms inspire of proton pump inhibitor (PPI) therapy, highlighting the need for multimodal diagnostics· [2]

**9. Risk Factors**

Key risk factors include obesity, hiatal hernia, male gender, and dietary triggers (e.g., caffeine, alcohol). Central obesity increases intra-abdominal pressure, compromising upper esophageal sphincter function.

**10. Clinical Presentation and Diagnosis**

Laryngopharyngeal reflux (LPR) presents differently from typical GERD symptoms such as heartburn, regurgitation, and esophagitis. However, approximately one-third of LPR patients also experience concurrent GERD. Studies indicate that LPR accounts for nearly 10% of otolaryngology clinic visits, with over half of patients complaining of hoarseness showing signs of reflux-related pathology. Due to its nonspecific and often subtle symptoms, LPR remains one of the most challenging conditions to diagnose accurately.

**11. Symptoms and Signs**

The most common clinical manifestations include:

1. Hoarseness (79%).
2. Chronic throat clearing (82%).
3. Globus sensation (88%).

Laryngoscopic observations frequently reveal:

1. Posterior commissure hypertrophy (70%).
2. Vocal fold edema (64%).
3. Diffuse laryngeal erythema.[3]

**12. Red Flags**

Symptoms such as dysphagia, unexplained weight loss, or asymmetrical laryngeal findings should prompt immediate evaluation to rule out malignancy.

| Laryngopharyngeal reflux | Gastro-oesophageal reflux disease |
|---|---|
| Involves upper oesophageal sphincter. | Involves lower oesophageal sphincter |
| Hoarseness, globus, dry cough, postnasal drip | Acid reflux, heartburn, chest pain. |
| Symptoms worse while upright position. | Symptoms worse while recumbent position. |
| No association with obesity/high body mass index (BMI). No history of deny heartburn, reflux. | Associated with obesity/high BMI Patients complain of heartburn and reflux. |

**13. Diagnostic Tools**

Reflux Symptom Index (RSI > 13)

The RSI is a 9-item questionnaire designed to evaluate symptoms associated with LPR. Each item is scored on a scale of 0 (no problem) to 5 (severe problem). A total score exceeding 13 suggests a high likelihood of LPR. The symptoms assessed include:

1. Hoarseness or voice issues (0–5)
2. Frequent throat clearing (0–5)
3. Excessive throat mucus or postnasal drip (0–5)
4. Difficulty swallowing (food, liquids, or pills) (0–5)
5. Coughing after meals or when lying down (0–5)
6. Breathing difficulties or choking episodes (0–5)
7. Persistent, bothersome cough (0–5)
8. Sensation of a lump or sticky feeling in the throat (0–5)
9. Heartburn or chest pain (0–5)

1.

Reflux Finding Score (RFS > 7)

The RFS is an 8-point endoscopic grading system used to identify laryngeal abnormalities linked to LPR.

A cumulative score above 7 indicates probable LPR. The assessed parameters include:

1. Subglottic edema (0 = absent, 2 = present)
2. Ventricular obliteration (2 = partial, 4 = complete)
3. Erythema/hyperemia (2 = localized to arytenoids, 4 = widespread)
4. Vocal fold edema (1 = mild, 2 = moderate, 3 = severe, 4 = polypoid)
5. Diffuse laryngeal edema (1 = mild, 2 = moderate, 3 = severe, 4 = obstructive)
6. Posterior commissure hypertrophy (1 = mild, 2 = moderate, 3 = severe, 4 = airway obstruction)
7. Granuloma/granulation tissue (0 = absent, 2 = present)
8. Thick mucus accumulation (0 = absent, 2 = present)

9. The maximum possible score is 26, with a threshold of 7 indicating LPR.

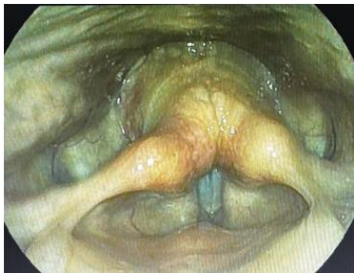

**Figure 1:** Normal Video laryngoscopy

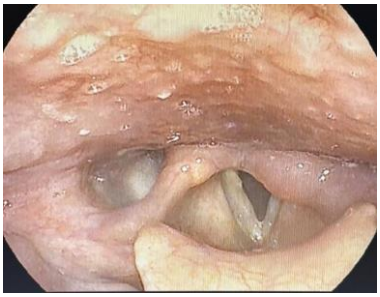

**Figure 2:** Left laryngeal edema

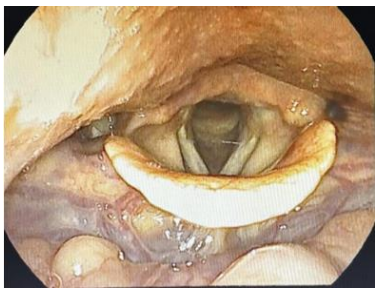

**Figure 3:** Granulations

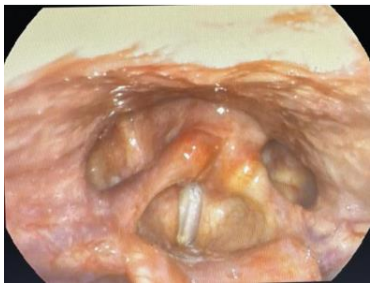

**Figure 4:** Erythema/Hyperemia of arytenoid

**14. Objective Diagnostic Evaluation for LPRD:**

Initial Laryngeal Evaluation:

Laryngoscopy: Mandatory for all patients with persistent dysphonia beyond 4 weeks (urgent evaluation recommended for smokers).

Laryngovideostroboscopy (LVS): Indicated when standard laryngoscopy appears normal; evaluates glottic closure patterns and mucosal wave dynamics.

**15. Endoscopic Procedures:**

Trans-Nasal Esophagoscopy (TNE):

1. Utilizes <6 mm endoscope.
2. Effective screening tool for established LPRD cases.

**16. Esophagogastroduodenoscopy (EGD):**

Performed with 10 mm endoscope under sedation.

Exminese ophagus, stomach, and proximal duodenum.
Note: Normal findings don't exclude LPRD diagnosis.

**17. Functional Assessments**
High-Resolution Esophageal Manometry:

1. Catheter-based motility study.
2. Detects esophageal dysmotility contributing to LPRD symptoms. [4]
3. Evaluates peristaltic coordination and bolus transit.

**18. pH Monitoring Systems**

1. Pharyngeal pH Measurement:
2. Well-tolerated with simplified scoring.
3. Limited by inability to predict PPI response.

**19. Hypopharyngeal-Esophageal Impedance with Dual pH Testing (Gold Standard)**

1. 24-hour monitoring using multichannel impedance-pH catheters.
2. Quantifies acid/non-acid reflux events to hypopharynx.
3. Correlates symptoms with reflux via patient-activated event marker.

**20. Biomarker Analysis**

Salivary Pepsin Assay (PEP-test):

1. Non-invasive, cost-effective screening.
2. 78% sensitivity, 65% specificity for LPRD.
3. Current role: Adjunctive diagnostic tool.

**21. Bile Acid Detection**

1. Mass spectrometry identifies duodenogastric reflux.
2. Salivary concentration >1 µmol/L correlates with LPR severity.
3. Predicts symptom intensity and mucosal damage.

**22. Voice Assessment**
Auditory Perceptual Evaluation:

1. Gold standard for voice disorder documentation.
2. Qualitatively assesses voice characteristics.
3. Quantifies degree of vocal deviation.

## 23. Treatment Strategies

Lifestyle and dietary modifications

First-line interventions: Elevate bedhead by 15 cm, avoid meals within 3 hours of bedtime.

Dietary changes: Alkaline diets (pH > 5) reduce pepsin activity. Zalvan et al. reported a 40% symptom reduction with Mediterranean diets compared to PPIs alone.

## 24. Pharmacotherapy

Proton pump inhibitors (PPIs): High-dose PPIs (e.g., omeprazole 40 mg BID) for 3–6 months are standard, though 40% of patients fail therapy due to non-acid reflux.

Alginates and magaldrate: Form protective barriers and are effective in mixed/non-acid reflux.

## 25. Surgical and Alternative Therapies

Fundoplication: Reserved for refractory cases, with 8090% success in GERD but variable outcomes in LPRD. Speech therapy: Addresses compensatory vocal behaviors.

## 26. Controversies and Challenges

PPI resistance: Linked to non-acid reflux or pepsin-mediated injury. Diagnostic variability: Lack of consensus on HEMII-pH thresholds.

Overlap with GERD: 30–50% of LPRD patients have concurrent GERD, necessitating dual management.

## 27. Future Directions

Personalized medicine: Tailoring treatment based on reflux phenotype (acid vs. non-acid). Novel therapies: Pepsin inhibitors (e.g., alginate-raft formulations) show promise.

Multidisciplinary care: Integrating ENT, gastroenterology, and speech therapy improves outcomes.

## 28. Conclusion

LPRD is a prevalent yet under recognized condition with significant quality-of-life implications. While empirical PPI therapy remains common, emerging diagnostic tools (e.g., HEMII-pH, pepsin assays) and multimodal treatments (diet, alginates, surgery) offer improved outcomes. Future research should focus on phenotyping patients and validating biomarkers to guide targeted therapy.

## 29. Source of Funding

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## 30. Conflict of Interest

None.

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