

LARYNGOPHARYNGEAL REFLUX— IT'S NOT GERD

Before you can alleviate symptoms in patients with LPR and ensure healing of the laryngopharynx, you must understand the difference between LPR and GERD.

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Laryngopharyngeal reflux (LPR) and gastro-esophageal reflux disease (GERD) are different disorders. The term LPR is used to describe the acid in the stomach that comes up into the throat at the level of the laryngopharynx. LPR causes irritation and changes in the larynx. GERD is caused by the backflow of gastric contents into the esophagus, which leads to tissue damage or esophagitis and heartburn.

indicate that more than 60 million adults suffer from heartburn at least once a month, and 25 million have daily heartburn.¹ Koufman and colleagues discovered that the prevalence of reflux in voice disorders may be as high as 60%.² Whether esophageal or laryngeal injury occurs is dependent upon the potency of the refluxate and the duration of the mucosal exposure.³

Distinguishing LPR from GERD

Patients with LPR have different symptoms and a different pathophysiologic mechanism than do patients with GERD. The most significant difference is that the majority of patients with LPR do not have esophagitis or heartburn. In numerous studies, the incidence of heartburn in patients with LPR is less than 40%, and the incidence of esophagitis is approximately 25%.⁴

Patients with LPR predominantly have daytime or upright reflux; those with GERD have nocturnal or supine reflux. Dysmotility and prolonged esophageal acid exposure occurs with GERD, while intermittent episodes of reflux are characteristics of LPR. Lower esophageal sphincter dysfunction is generally present with GERD, whereas patients with LPR are likely to have upper esophageal sphincter dysfunction.³ The larynx also differs from the esophagus in its response to reflux in that the larynx lacks the extrinsic and intrinsic epithelial defenses present in the esophagus. The mucosa of the larynx is thin and fragile and cannot protect itself against gastric acids.^{3,5} These differences in mechanism account for the differences in symptoms and response to treatment.

Clinical presentation

The most common symptom of LPR is reflux laryngitis: Hoarseness is found in 71% of affected patients.³ Forty-seven percent of patients complain of having globus pharyngeus (a lump in the throat), and 51% demonstrate cough,³ chronic throat clearing, dysphagia, and sore throat are also common symptoms.³ Reflux laryngitis is characterized by hoarse vocalization, voice fatigue, and breaking of the voice.⁶ Reflux has also been associated with vocal cord polyps, vocal cord granulomas, laryngospasm, and paradoxical vocal fold movement. LPR was found in 58% of patients with laryngeal carcinoma and in 56% of those with subglottic stenosis.³

Other manifestations of LPR may include asthma and sinusitis.^{3,4,6,7} Reflux is often overlooked as a factor that exacerbates asthma. Research has shown that when stomach acid is successfully suppressed, 73% of patients with asthma have improvements in asthma symptoms and pulmonary function.⁷ Patients with LPR frequently complain of chronic throat clearing,

In This Article

Key Points

- LPR and GERD are different disorders causing different symptoms and signs.
- A workup for LPR is needed in patients with chronic or intermittent hoarseness, chronic throat clearing, chronic cough, voice fatigue or changes, globus sensation, and sore throat.
- The diagnosis of LPR is made by the history, visualization, and using indicated studies.
- Treatment is generally twice daily dosing of a PPI for a minimum of 6 months.

Competencies

Medical Knowledge	◆◆◆◆
Interpersonal & communication Skills	◆◆◆
Patient Care	◆◆◆◆
Professionalism	◆◆
Practice-based learning and improvement	◆◆
Systems-based practice	◆◆

Reflux literally means backflow. It is derived from the Latin words re, meaning back, and fluere, meaning to flow. Everyone experiences physiological reflux to some degree after eating. Recent surveys of Americans

excessive mucus in the throat, and postnasal drip. These symptoms come from the mucosal irritation associated with LPR --- most patients do not have sinus disease.^{4,6} These common symptoms can complicate the diagnosis of LPR and delay appropriate treatment.

Diagnosis

LPR is diagnosed based on clinical symptoms and laryngeal findings. A detailed history of symptoms will aid the evaluation.

- Ask about changes in the patient's voice and the character and duration of these changes. Are the changes intermittent, and do they occur late in the day? If they are and do, this suggests LPR.
- Patients who describe a sensation of a lump in their throat with chronic throat clearing have signs of LPR. Excessive throat mucus that is discolored may be gastric contents and not a sign of sinus disease.
- Include questions about voice misuse or abuse, such as might occur in patients who must speak continuously and loudly over noise at their workplace. Patients who misuse or abuse their voice can have changes in it unrelated to LPR.
- Discuss any heartburn the patient may have and any remedies the patient has used.
- Be sure to ask about use of tobacco or alcohol, and obtain quantities. Studies indicate an established correlation between smoking, alcohol consumption, and cancer and between reflux and cancer. In one large study, 92% of patients with GERD were found to be smokers. Use of tobacco and alcohol decreases lower esophageal sphincter pressure, impairs mucosal resistance, delays gastric emptying, and stimulates gastric hypersecretion. Of patients with laryngeal carcinoma who were lifelong nonsmokers, all had reflux symptoms and abnormal results on barium esophagography, suggesting that reflux is a cofactor in the development of upper digestive system carcinomas.³
- Dysphagia should be addressed: A patient who has changes in swallowing with liquid or food bolus may have an esophageal lesion or cancer and should have a further workup.
- Establish whether the patient has any respiratory problems associated with voice changes.⁶ Laryngeal spasm or paradoxical vocal fold movements may suggest LPR as an underlying disorder.

Flexible laryngoscopy or mirror examination by an otolaryngologist is recommended for patients whose history suggests LPR. These procedures are easily done in the office at the initial visit.

Patients will have erythema of the arytenoids and posterior commissure, which may extend to the true and false vocal cords, subglottic mucosa, and pharyngeal mucosa. The interarytenoid space will exhibit mucosal hypertrophy. In advanced cases, the posterior commissure will have hyperkeratotic epithelium.

Vocal cord granulomas occur at the vocal process at the arytenoid cartilage. Because of the posterior position of the vocal process, they are greatly influenced by reflux. Vocal cord nodules are likely a result of untreated LPR in an adult and are seen bilaterally at the junction of the anterior and middle thirds of the phonating surface of the true vocal cords.

Laryngeal stenosis is a severe consequence of ongoing LPR and may be life-threatening. Affected patients generally have a traumatized larynx, most often from direct trauma or intubation injury. Seventy-five percent of patients with diagnosed laryngeal and tracheal stenosis demonstrate significant reflux.⁸

Videostroboscopy is a video examination of the larynx using a rigid fiberoptic endoscope with an intermittent xenon light source that is activated by vocal fold movement. The video examination allows enlarged, slow-motion visualization of the vocal cords. Using playback, the clinician can study and document vibratory motion and laryngeal problems that cannot otherwise be appreciated. This technology is available for use in an office setting without anesthesia.⁹ Direct visualization of the vocal cords also enhances patient education. An acoustic voice evaluation, performed by a speech therapist, often adds a collaborative approach to the videostroboscopy.

Barium esophagography is for patients who are suspected of having GERD or LPR. This radiographic study will identify motility and peristaltic abnormalities, esophageal lesions, spontaneous reflux, hiatal hernia, and lower esophageal sphincter disorders. Although barium esophagoscopy is less sensitive in diagnosing LPR, it is advisable for a complete workup when treatment fails or it is otherwise clinically indicated, such as in patients who have signs of GERD, esophageal dysfunction, or an unclear diagnosis.

Ambulatory pH monitoring has been used extensively in the stuffy of GERD and LPR. This test is invasive and uncomfortable for most patients. At present, it is not routinely used in the initial evaluation of LPR, but it may be useful in the diagnosis of laryngeal stenosis and paroxysmal laryngeal spasm or for patients whose symptoms do not resolve with treatment. If the patient

continues to have symptoms of LPR but the findings from the initial workup are negative, ambulatory, 24-hour, double-probe pH monitoring is till the gold standard for the diagnosis.

Direct laryngoscopy (and suspension microlaryngoscopy, which is the same procedure using different equipment) is performed under general anesthesia, usually by an otolaryngologist. This method allows superior or visualization of the larynx and surrounding structures, and it also permits biopsy of vocal cord lesions.

Treatment

Proton pump inhibitors (PPIs) and H₂-receptor antagonists are the mainstay of treatment for LPR as well as GERD. Treatment of LPR depends on severity of symptoms, but in general LPR requires a more aggressive, longer course of therapy than does GERD. Mild symptoms may be treated with H₂-receptor antagonists and dietary and lifestyle modifications. Patient education regarding precautions to prevent reflux is the key to success. Half of patients with mild LPR will have resolution of symptoms with the indicated lifestyle changes.¹⁰ Most patients with moderate to severe LPR on examination will require at least twice daily dosing with a PPI because none of the PPIs suppresses acid for more than 16.8 hours¹ and laryngeal damage may occur with as few as three intermittent LPR episodes a week.^{3,4} Most patients will report significant improvement in symptoms within 2 to 3 months after treatment is initiated; however, studies show that healing takes 6 months or longer.^{4,5} Twice daily dosing of a PPI for a minimum of 6 months is recommended to reverse tissue injury in the larynx. Approximately 20% to 30% of patients do not respond to the standard regimen of twice-daily dosing with a PPI.^{3,11} A subgroup of patients may require prolonged or lifetime treatment if their symptoms recur when they stop the medication. Follow-up examinations should be scheduled every 3 months to monitor symptoms or

more often if the disease is not responding to treatment or the patient has new symptoms. Patients with persistent symptoms and laryngeal changes should undergo further workup.

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