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EDITORIAL

Dear colleagues,

Warm regards and greetings from the editor's desk. Laryngology per se has become an independent medical specialty under the broader entity of otorhinolaryngology. In-depth knowledge of disease pathophysiology has further led to the emergence of subspecialties like phonology, deglutology and airway surgery. In spite of all these recent advancements some clinical conditions still baffle us today in terms of management and treatment protocol, namely Laryngopharyngeal reflux disease (LPRD).

The LPRD refers to an inflammatory reaction of the mucous membrane of pharynx, larynx and other associated respiratory organs, caused by a reflux of stomach contents into the esophagus. Although the terminology "LPR" was recognized by some otolaryngologists way back in the late 1960s, international researchers accepted it as an extraesophageal manifestation of gastroesophageal reflux disorder (GERD) only in the late 80s and 90s. The term "LPR" was coined and popularized by Koufmann in 1991. As the food bolus is propelled from the oropharynx to the esophagus the epiglottis closes the laryngeal inlet to prevent aspirations, the upper esophageal sphincter (UES) relaxes and the bolus enters the esophagus which gradually reaches its lower end through involuntary peristaltic contractions. At the lower end the lower esophageal sphincter (LES) and the diaphragm relax to let the bolus enter the stomach. The UES and LES contract rhythmically even at rest due to continuous neural activity. The LES relaxation can occur for 10–60 sec even in the absence of peristalsis and is known as transient LES relaxation (TLESR). This phenomenon is vagus mediated and is the primary cause for GER in adults. The GER is a physiological phenomenon where even a healthy individual can have multiple episodes of reflux in a day. It can be labeled as GERD only when an individual is symptomatic and there are demonstrable changes in the lower end of esophagus in upper GI endoscopy.

The LPRD patients usually present with hoarseness, globus sensation, excessive throat clearing, postnasal drip, chronic dry cough, choking sensation, dysphagia and even heartburn. Smoking and alcoholism are frequent associations of LPRD–GERD patients usually have their complaints only at night during lying down whereas symptoms of LPRD are experienced even in erect posture during daytime. Fiberoptic laryngoscopy reveals thick mucus, congested laryngeal inlet, edematous vocal folds, interarytenoid pachydermia and pseudosulcus. Reflux symptom index (RSI), a self-assessment score and reflux finding score (RFS), based on laryngoscopic findings, are two diagnostic tools designed by Belafsky. An RSI score of more than 10 and an RFS score of more than 7 suggest LPRD. Upper GI endoscopy and 24-hour pH monitoring are other diagnostic tools to evaluate the presence of GERD. Studies have revealed that LPRD plays some role in upper aerodigestive tract malignancies.

The mainstay of treating LPRD is lifestyle modifications and medications. Lifestyle modification encompasses avoidance of smoking, alcohol, spicy and fatty foods, aerated drinks and caffeine. Small frequent meals, avoiding heavy meals just before bedtime, weight reduction, avoiding tight fitting clothes and elevating the head end by 15 degrees are other proven factors. Medications include long-term proton pump inhibitors, usually double dose, along with H_2 blockers at bedtime (for 12 weeks) to neutralize the gastric acid. Alginates after food for many months together help combat the pepsin factor in managing LPRD. Association of hiatus hernias and resistant cases may require surgical intervention.

Reassurance plays a key role in managing these patients and the benefit of good counseling is worth mentioning. The LPRD patients should be treated for months together until they are symptom free and resistant cases should be followed up closely.

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