CLINICAL VIGNETTE

Laryngopharyngeal Reflux: a Syndrome that Mimics Asthma

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A 45-year-old female with no history of childhood asthma or seasonal allergies presented with history of chronic cough, intermittent wheezing, and dysphonia of four years duration. She was a lifelong nonsmoker. She was evaluated by her primary care physician three years ago for these symptoms and a presumptive diagnosis of asthma was made. No spirometry was done at that time. Treatment with twice daily combination regimen of high-dose inhaled steroid and a long acting beta agonist was instituted with partial relief of symptoms. In the two years preceding her evaluation at our clinic, she had suffered from recurrent episodes of bronchitis treated with short courses of antibiotic regimen. She was referred to our clinic for further evaluation of asthma that was considered difficult to control.

She denied any family history of asthma. She had no pets at home and was an elementary school teacher by profession.

Physical examination revealed a woman in no respiratory distress. Abnormal findings included inspiratory and expiratory wheezes that were heard diffusely but were loudest over the central airways.

CXR was done with normal lung parenchyma. Spirometry demonstrated normal spriometric indices but with mild flattened inspiratory loop on the flow volume curve. There was no improvement after bronchodilator administration. For further evaluation of an abnormal inspiratory flow volume loop, she was referred to an ENT physician. She underwent a flexible laryngoscopy and stroboscopy, which identified severe edema of the true and false vocal cords, consistent with laryngopharyngeal reflux.

A methacholine challenge test was performed for questionable diagnosis of asthma, which identified no change in FEV1, even with the highest dose of methacholine.

She was taken off all inhaler regimens and started on a twice daily regimen of proton pump inhibitor medications. She was also placed on strict dietary restrictions for reflux and was advised to modify her lifestyle to minimize reflux.

At 6 months follow-up, she reported a complete resolution of cough, wheezing, and dysphonia. At 1 year follow-up, she reported of no further episodes of bronchitis.

Discussion

Laryngopharyngeal reflux (LPR) is the retrograde movement of gastric contents (acid and enzymes such as pepsin) into the larynx, pharynx, trachea, and bronchus. Its clinical manifestation and symptoms differ distinctly from the typical symptoms of GERD. The most frequent typical symptom of GERD is heartburn, which occurs in at least 75% of patients. In contrast, LPR manifests with symptoms such as hoarseness, globus pharyngeus, chronic cough, intermittent wheezing or choking episodes, constant throat clearing, and/or mild dysphagia. Most patients are relatively unaware of LPR with only about 35% reporting heartburn. The reason for this is that refluxate spends very little time in the esophagus and does most of its damage in the larynx. These symptoms can be easily misdiagnosed as asthma, as in our patient. In two different surveys, when multiple PCP were given a questionnaire about LPR, it was concluded that the majority of them were not acquainted with LPR nor the appropriate treatment for LPR.²⁻³

Diagnosing LPR can be very challenging as symptoms can be very nonspecific. A reflux symptom index (RSI) (Table1) is a nine item validated questionnaire that be used to establish the initial diagnosis of LPR and monitor response to treatment. An RSI greater than 13 strongly suggests LPR. The patient can then be referred to ENT specialist for a transnasal fiberoptic laryngoscopy, which is an essential tool in the evaluation of patients with suspected LPR. The endoscopic laryngeal findings attributable to reflux have been well-described and include erythema and edema of the posterior commissure and arytenoid cartilages, vocal fold granulomas, nodules, polyps, and possibly carcinoma. ⁴⁻⁵ A persistently abnormal inspiratory curve in the presence of otherwise normal spirometry can be suggestive of glottic disorders related to reflux.⁶ Another tool utilized to confirm the diagnosis of LPR and assessing the magnitude of the problem is dual probe 24 hour pH monitoring, during which probes are placed simultaneously in the pharynx and esophagus.⁷⁻⁹ An immunologic pepsin assay (Peptest) is a newer diagnostic technique, which has been shown to be a rapid, sensitive, and specific tool for detecting LPR. 10 A new pH pharyngeal catheter, manufactured by Restech in San Diego, CA, USA, was found to be highly sensitive and minimally invasive device for the detection of liquid or vapors of acid reflux in the posterior oropharynx. 11

The laryngopharyngeal epithelium is far more susceptible to reflux-related tissue injury than the esophageal epithelium. Because of these differences, treatment algorithms for LPR and GERD are very different. Medical therapy for LPR usually entails higher doses of proton pump inhibitors, which are more potent than antacids and H2 blockers over an extended period of time (3 months or longer). 12-13 This is usually combined with behavior modifications that include weight reduction, avoidance of food high in fat and caffeine, and elevation of the head of the bed. Surgical options for treating LPR are usually reserved for medical treatment failures as well as younger individuals with severe LPR who face a lifetime of expensive medical therapy with unknown long-term consequence. Surgical options include minimally invasive procedures such as radiofrequency ablation of the lower esophageal sphincter as well as endoscopic Nissen Fundoplication, a procedure where the surgeon wraps the stomach around the distal esophagus to create a tight valve. It has proven to be very successful in preventing reflux into the esophagus as well as the pharynx (throat) and larynx. 14

It is important to remember that LPR can mimic or co-exist with asthma. If not correctly diagnosed, it can lead to overtreatment with corticosteroids with consequent morbidity, including increase susceptibility to respiratory tract infections, such as in our patient. Once inhaled steroids were discontinued, no further episodes of bronchitis occurred by her two year follow-up visit.

Figures

 Table 1. Reflux Symptom Index

Reflux Symptom Index

0 = no proble	m, 5 = severe proble
1. Hoarseness or a problem with your voice	0 1 2 3 4 5
2. Clearing your throat	0 1 2 3 4 5
3. Excess throat mucous or postnasal drip	0 1 2 3 4 5
4. Difficulty swallowing food, liquids, or pills	0 1 2 3 4 5
5. Coughing after you ate or after lying down	0 1 2 3 4 5
6. Breathing difficulties or choking episodes	0 1 2 3 4 5
7. Troublesome or annoying cough	0 1 2 3 4 5
Sensations of something sticking in your throat or a lump in your throat	0 1 2 3 4 5
9. Heartburn, chest pain, indigestion, or stomach acid coming up	0 1 2 3 4 5
	Total

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