CASE REPORT

THE PATIENT

60-year-old man

SIGNS & SYMPTOMS

- Chronic cough
- History of GERD & dyslipidemia
- Throat tickle

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THE CASE

A 60-year-old man with a past medical history of gastroesophageal reflux disease (GERD) and dyslipidemia presented to his family physician for evaluation of chronic cough. Five years prior, the patient had developed a high fever and respiratory symptoms, including a cough, and was believed to have had severe otitis media. He was treated with multiple courses of antibiotics and corticosteroids for persistent otitis media. Although the condition eventually resolved, his cough continued.

The persistent cough prompted the patient to consult a succession of specialists. First, he saw a gastroenterologist; following an esophagogastroduodenoscopy, he was prescribed pantoprazole. Despite the proton-pump inhibitor (PPI) therapy, the cough remained. Next, he had multiple visits with an otolaryngologist but that yielded no specific diagnosis for the cough. He also saw an allergist-immunologist, who identified a ragweed allergy, gave him a diagnosis of cough-variant asthma, and prescribed antihistamines and mometasone furoate and formoterol fumarate dihydrate. Neither was helpful.

After 5 years of frustration, the patient complained to his family physician that he still had a cough and "a tickle" in his throat that was worsened by speaking and drinking cold beverages. He denied fever, shortness of breath, nausea, vomiting, or any other associated symptoms.

THE DIAGNOSIS

The failed treatment attempts with antihistamines, corticosteroids, bronchodilators, and PPI therapy excluded multiple etiologies for the cough. The throat discomfort and feeling of a "tickle" prompted us to consider a nerve-related disorder on the differential. The diagnosis of laryngeal sensory neuropathy (LSN) was considered.

DISCUSSION

LSN is a relatively uncommon cause of chronic refractory cough that can also manifest with throat discomfort, dysphagia, and dysphonia.[1] It is thought to result from some type of insult to the recurrent laryngeal nerve or superior laryngeal nerve via viral infections, metabolic changes, or mechanical trauma, leading to a change in the firing threshold.[2] The hypothesis of nerve damage is supported by the increased incidence of LSN in patients with goiters and those with type 2 diabetes.[3,4] When there is a decrease in the laryngeal sensory threshold, dysfunctional laryngeal behavior results, leading to symptoms such as persistent cough and throat clearing.

Diagnosis. LSN is often diagnosed clinically, after GERD, allergies, asthma, angiotensin-converting enzyme inhibitor intake, and psychogenic disorders have been ruled out.[1]

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The throat discomfort and feeling of a "tickle" prompted us to consider a nerve-related disorder on the differential.

Our patient had a prior diagnosis or investigation of nearly all of these conditions. Other clues pointing to an LSN diagnosis include a cough lasting 8 weeks or more, recurrent sensory disturbances (such as a tickle) of instantaneous onset before each cough episode, triggers that can include talking or a change in air temperature, daily coughing episodes numbering in the 10s to 100s, and a nonproductive cough.[5,6]

Beyond clinical clues, laryngeal electromyography, which evaluates the neuromuscular system in the larynx by recording action potentials generated in the laryngeal muscles during contraction, can be used for diagnosis.[4] Videostroboscopy, which allows for an enlarged and slow motion view of the vocal cords, can also be used.

Treatment. To both confirm the diagnosis and treat the patient in a rapid, practical fashion, a trial of a neuromodulating agent such as pregabalin or gabapentin can be employed.[6-9] A study identifying 28 LSN patients found symptomatic relief in 68% of patients taking gabapentin 100 to 900 mg/d.[2] In another study, 12 LSN patients given pregabalin found relief after a 1-month regimen.[1] Another study of 12 patients showed amitriptyline hydrochloride and gabapentin provided a positive response in 2 months, and the addition of reflux precautions and acid-suppression therapy was helpful.[9] Finally, a group of 32 patients trialed on 3 different medications (amitriptyline, desipramine, and gabapentin) found similar efficacy among the 3.[6]

Another option. Aside from medications, botulinum toxin type A has been shown in a case series to directly decrease laryngeal hypertonicity and possibly reduce neurogenic inflammation and neuropeptide-mediated cough.[10] Another study found that 18 patients with neurogenic cough who received superior laryngeal nerve blocks had cough severity index scores decrease from an average of 26.8 pretreatment to 14.6 posttreatment ($P<.0001$).[11]

Our patient agreed to a trial of gabapentin 300 mg once a day, with titration up to a maximum of 900 mg tid. When the patient returned to the clinic 4 months later, he reported that when he reached 300 mg bid, the cough completely resolved.

THE TAKEAWAY

A persistent cough with minimal identifiable triggers is a huge disruption to a patient's life; having to visit multiple specialists before receiving a diagnosis compounds that. In our patient's case, the process took 5 years, which underscores how important it is that LSN be considered in the differential diagnosis. Since this is generally a diagnosis of exclusion, it is important to take a careful history of a patient with a chronic cough. If LSN seems likely, trialing a patient on neuromodulating medication is the next best step, with dose titration if necessary. JFP

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