Hoarseness and cough in a 67-year-old woman

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A 67-year-old white woman presented with an approximately 1-month history of hoarseness and nonproductive cough. She had quit smoking 20 years earlier and denied chest pain and dysphagia. She was referred to an otorhinolaryngologist for further assessment. A computed tomography (CT) scan of the neck and chest was then requested.

Contrast-enhanced CT images of the neck and chest are displayed *(Figures 1–4)*. In addition, a positron emission tomography scan (not shown) revealed increased uptake in the aorticopulmonary window lymph node and the left lung nodule.

Figure 1. Axial postcontrast CT image demonstrates an enlarged lymph node complex (arrow) in the aorticopulmonary window.

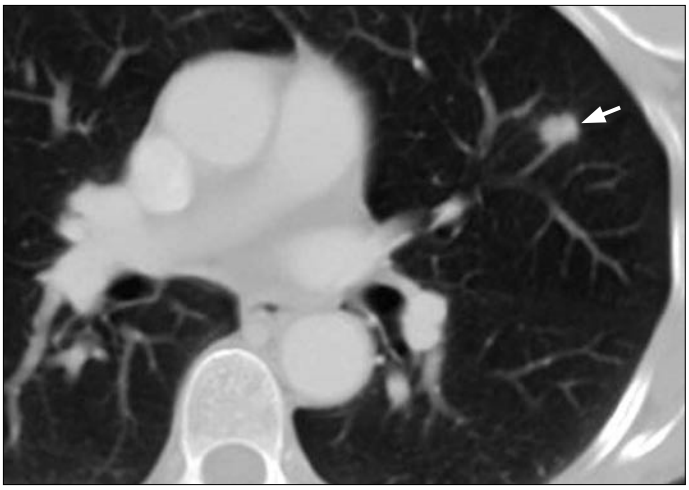

Figure 2. Axial postcontrast CT image shows a nodule (arrow) in the upper lobe of the left lung.

A thoracic surgeon was consulted for fiber-optic bronchoscopy and mediastinoscopy. On bronchoscopic evaluation, only mild atrophy of the bronchial mucosa with no endobronchial mass was visualized. Further examination with a mediastinoscope revealed a solid and immobile lymph node complex in the aorticopulmonary window. Multiple biopsies of this lymph node complex were obtained.

What is the most likely diagnosis?

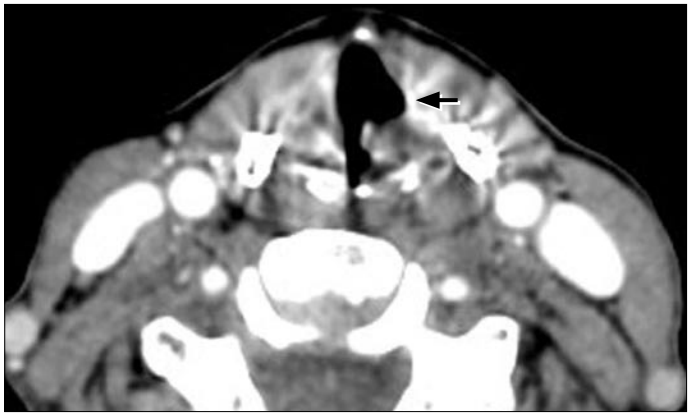

Figure 3. Axial CT image through the larynx reveals a dilated left laryngeal ventricle (arrow).

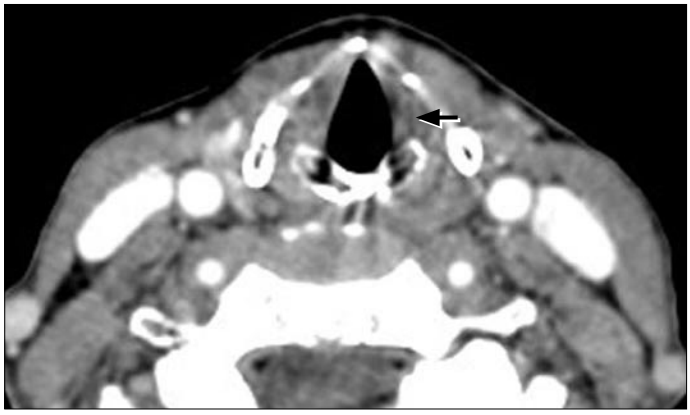

Figure 4. Axial CT image just inferior to the dilated laryngeal ventricle demonstrates the reduced size of the left thyroarytenoid muscle, or true vocal cord, with decreased density (arrow).

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DIAGNOSIS: Left vocal cord paralysis secondary to malignant invasion of the left recurrent laryngeal nerve within the mediastinum.

Pathology of the lymph node specimen revealed poorly differentiated large-cell carcinoma consistent with lung origin. The patient did well postoperatively and will follow up with an oncologist to discuss treatment options.

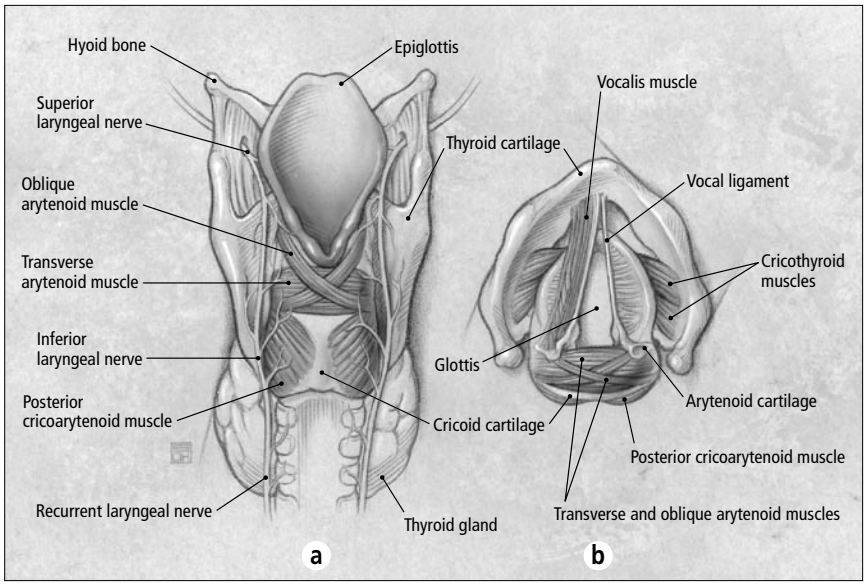

Figure 6. Major muscles and cartilages of the larynx. **(a)** Posterior laryngeal view. **(b)** Cross-sectional view of the larynx, looking from overhead. Reprinted with permission of Steve Oh, KO Studios, ©1998.

DISCUSSION

Unilateral vocal cord paralysis occurs secondary to dysfunction of the recurrent laryngeal or vagus nerve innervating the larynx. The patient typically presents with a fairly sudden onset of a "breathy" voice because of glottal incompetence resulting from incomplete adduction of the paralyzed vocal cord. In addition, the patient may complain of his or her "voice going away" and shortness of breath. Normally, upon speaking, both vocal cords adduct to allow for glottal closure and subsequent induction of vocal cord vibration (1).

The etiology of unilateral vocal cord paralysis can be categorized into 3 main groups: surgical iatrogenic injuries, malignant invasion, and idiopathic paralysis. Surgical iatrogenic injuries may be a complication of thyroidectomy, carotid endarterectomy, anterior cervical disc surgery, and thoracic or mediastinal surgery. Malignant invasion of the vagus or recurrent laryngeal nerves can also occur with skull base tumors, thyroid neoplasms, carcinoma of the lung, esophageal carcinoma, and metastases to the mediastinum (well illustrated in this case). A third etiology is idiopathic—when a definite etiology for the paralysis cannot be determined. These cases may be attributed to a recent viral infection or inflammatory process (1).

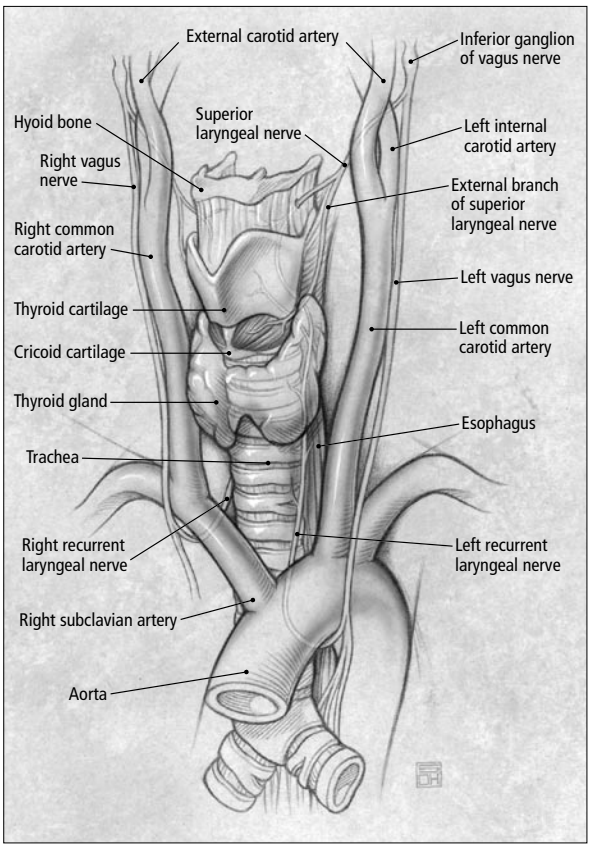

Figure 5. Courses of both recurrent laryngeal nerves and the neighboring structures. Reprinted with permission of Steve Oh, KO Studios, ©1998.

A physician should begin investigating vocal cord paralysis by following the route of the vagus and recurrent laryngeal nerve on CT scans from the skull base to the superior mediastinum. Understanding the anatomy of both vagus and recurrent laryngeal nerves is essential. The vagus nerve originates in the brain stem, specifically the nucleus ambiguus of the medulla. The vagus nerve then travels along the carotid sheath with the jugular vein and internal carotid artery (2).

The left vagus nerve gives rise to the left recurrent laryngeal nerve as the vagus nerve descends just lateral and anterior to the arch of the aorta. The left recurrent laryngeal nerve then loops under the aortic arch adjacent to the ligamentum arteriosum and ascends in the tracheoesophageal groove until penetrating the larynx, supplying the intrinsic muscles of the larynx (2). In contrast, the right recurrent laryngeal nerve loops under the right subclavian artery before ascending to innervate the larynx (*Figure 5*).

In addition to knowing the pathway of both recurrent laryngeal nerves, the physician must be aware that, superior to the hyoid bone, the vagus nerves have small branches to the pharynx, which assist with swallowing and the gag reflex. Therefore, if pharyngeal muscle atrophy or a deviated uvula is found on physical examination or imaging studies, vagus nerve lesions high in the neck or in the skull base may be present. In this scenario, gadolinium-enhanced magnetic resonance imaging (MRI) is the preferred modality because of its superior separation of soft tissues in this region (3).

If after obtaining a focused history and performing a head and neck examination, which should include visualization of the larynx, the physician suspects unilateral vocal cord paralysis, then imaging studies are recommended. The posteroanterior and

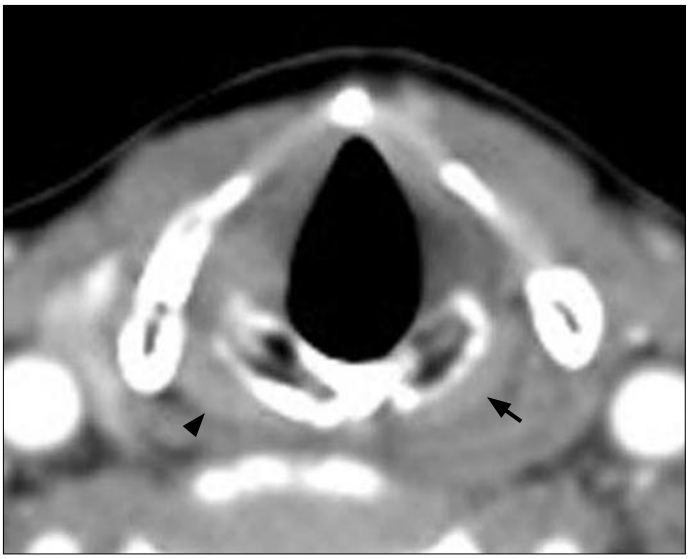

Figure 7. Axial CT image at the cricoid level demonstrates mild atrophy of the left posterior cricoarytenoid muscle (arrow) compared with the normal right side (arrowhead).

Figure 8. Coronal CT image of the neck illustrates a dilated left laryngeal ventricle (star). The laryngeal ventricle is the space between the false vocal cord (arrow) and true vocal cord (arrowhead).

lateral chest radiograph is the first screening examination for a patient with unilateral vocal cord paralysis of unknown etiology. A chest malignancy such as Pancoast tumor or mediastinal mass may be revealed. However, the ordering physician needs to be aware that >50% of mediastinal and lung masses detected on CT are not visualized on plain radiographs (3). For this reason, some physicians move directly to ordering a CT or MRI, which includes the entire path of the vagus and recurrent laryngeal nerves involved.

The larynx is an intricate structure with multiple anatomic parts that work together during voice production. The laryngeal anatomy consists of 4 fundamental anatomic components: a cartilaginous skeleton, intrinsic muscles, extrinsic muscles, and a mucosal lining *(Figure 6)*. The cartilaginous skeleton contains the vocal cords (thyroarytenoid muscles) and comprises the thyroid, cricoid, and arytenoid cartilages. The extrinsic muscles connect

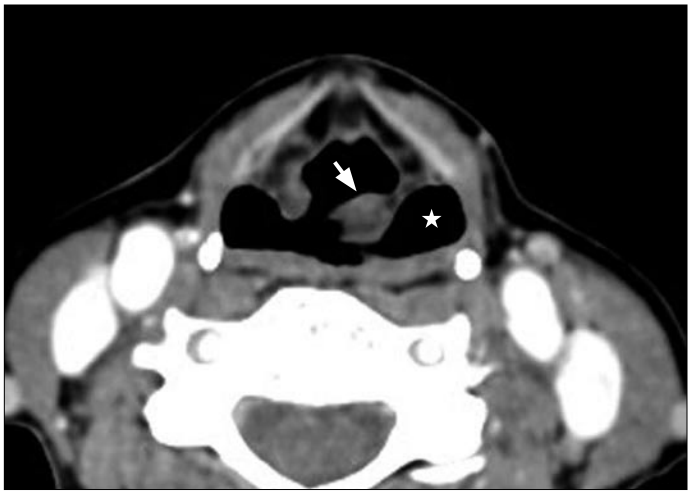

Figure 9. Axial CT image through the superior larynx reveals an anteromedial rotation of the left aryepiglottic fold (arrow) with resultant mild dilatation of the left pyriform sinus (star).

these cartilages with other structures in the head and neck. The intrinsic muscles are responsible for changing the position and tension of the vocal cords. With the exception of the cricothyroid muscle, which is innervated by the superior laryngeal nerve, the recurrent laryngeal nerve innervates all of the intrinsic laryngeal muscles. Overall, voice production requires 2 essential components: airflow generation and vocal cord vibration, which creates and shapes sound (4).

The specific imaging features of vocal cord paralysis on CT or MRI include atrophy of the thyroarytenoid muscle, anteromedial deviation of the arytenoid cartilage, enlarged laryngeal ventricle, enlarged pyriform sinus, and a paramedian or intermediate vocal cord. Recently, it has also been discovered that atrophy of the posterior cricoarytenoid muscle, an intrinsic muscle of the larynx, occurs with vocal cord paralysis (5). Most of these findings are secondary to atrophy of the thyroarytenoid muscle, which makes up the bulk of the true vocal cord. As the muscle decreases in size, the vocal cord becomes thinner and more pointed in the coronal plane. In addition, with atrophy of the thyroarytenoid muscle, the laryngeal ventricle enlarges. Lastly, with vocal cord paralysis, the ipsilateral aryepiglottic fold moves medially, secondarily enlarging the ipsilateral pyriform sinus (6). These imaging findings are well illustrated in this patient *(Figures 3, 4, 7, 8,* and *9)*.

Various treatments are available for unilateral vocal cord paralysis. The 2 main approaches are voice therapy and surgical therapy. Voice therapy is used primarily when the paralyzed vocal cord is in a favorable position or when the patient is unable to have surgery because of medical limitations. The most common voice therapy techniques involve avoidance of irritants such as tobacco and alcohol, proper use of respiratory support for phonation, phrase and word timing, and pitch alteration. Surgical procedures aim to medialize the paralyzed vocal cord. Temporary treatment utilizes the Gelfoam vocal cord injection, which effectively medializes the cord for 4 to 12 weeks. Two options are available for permanent surgical therapy. Fat injection of the vocal cord (lipoinjection) involves collecting a small amount of the patient's fat—usually via liposuction or open incision in the abdomen—and injecting it into the deep portion of the vocal cord. A more advanced surgical method, known as laryngeal

framework surgery, involves manipulating the paralyzed vocal cord through an external approach and repositioning the arytenoid cartilage (7).

CONCLUSION

Unilateral vocal cord paralysis is a problem frequently encountered in clinical practice. The evaluation of patients with vocal cord paralysis has evolved dramatically over the last couple of decades with precise imaging and advanced physical examination techniques. Unilateral vocal cord paralysis is most often a manifestation of other pathologic findings, and the examination requires meticulous inspection along the entire path of the vagus and recurrent laryngeal nerves.

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