Unusual paratracheal masses presenting with vocal fold paralysis

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Abstract
Most paratracheal masses are of thyroid origin. We describe two cases of vocal fold paralysis that were caused by unusual paratracheal masses. In one case, a 35-year-old man was found to have a malignant lymphoma that originated in the mediastinum and extended above the clavicle. The other patient was a 53-year-old man with an enlarged left thyroid lobe, tumor invasion of the adjacent larynx and trachea, and multiple pulmonary nodules all due to adenoid cystic carcinoma. Unusual paratracheal masses presenting with vocal fold paralysis may mimic thyroid malignancies, thereby posing both diagnostic and therapeutic challenges. Fine-needle aspiration cytology is often helpful in making a definitive diagnosis, but incisional biopsy is necessary in some cases.

Introduction
Most lesions in the paratracheal region are of thyroid origin. The presence of vocal fold paralysis in a patient presenting with a thyroid mass is generally believed to suggest malignancy. A variety of nonthyroid lesions may arise in the paratracheal position and thus mimic thyroid neoplasms, but they rarely occur with vocal fold paralysis. For this reason, physicians may unnecessarily limit their differential diagnosis when evaluating patients with a paratracheal mass and vocal fold paralysis. This failure to consider an extrathyroidal origin might misdirect their diagnostic and therapeutic strategies and lead to delay in treatment and an increase in morbidity. In this article, we describe two unusual paratracheal masses in patients who presented with vocal fold paralysis. We discuss the broad differential diagnosis of lesions presenting in this manner, and we suggest a simple diagnostic algorithm.

Case reports
We retrospectively reviewed the medical records, including radiologic images and pathology specimens, of 2 patients who had previously presented to the Department of Otolaryngology–Head and Neck Surgery at Virginia Commonwealth University with paratracheal masses that had caused vocal fold paralysis. Our review was approved by the university’s Office of Research Subjects Protection.

Patient 1. An otherwise healthy 35-year-old man presented with a 2-month history of hoarseness and a left lower neck mass. He denied hemoptysis, dysphagia, and dyspnea. He had a 10-pack-year history of cigarette use, but he had stopped smoking 2 years earlier. The physical examination was notable for a raspy voice with obvious diplophonia and mild inspiratory stridor. Indirect laryngoscopy revealed that the left vocal fold was in the paramedian position and immobile. External compression of the airway was visible at the immediate subglottis on the left. Palpation of the neck detected a 3- to 4-cm firm, nontender, left paratracheal mass that elevated with swallowing; the mass extended below the clavicle. The cervical trachea was deviated 2 cm to the right of midline.

Prior to the patient’s scheduled neck computed tomography (CT) and follow-up, he presented to the emergency department with hemoptysis and a new chest mass that was palpable between the first and second ribs. Chest CT detected a 10 × 11-cm homogeneous upper mediastinal mass that extended above the left clavicle (figure 1, A). The trachea, which remained deviated to the right, was compressed. The mass encircled the aortic arch and pulmonary artery and extended to the cardiac base. Fine-needle aspiration cytology (FNAC) showed predominantly spindle cells with scattered lymphocytes (figure 1, B), suggesting a differential diagnosis that included thymoma, spindle cell carcinoma, spindle cell variants of medullary and anaplastic thyroid carcinomas, and a spindle cell variant of lymphoma.

The patient underwent airway endoscopy and transcervical incisional biopsy of the mass. Laryngoscopy confirmed that the trachea was deviated to the right and compressed by approximately 50%, but no intraluminal invasion was seen. Pathology revealed a diffuse, large B cell lymphoma, and tumor cells were positive for CD10,
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CD19, CD20, CD45, HLA-DR, and lambda light chain on immunohistochemistry and flow cytometry (figure 1, C). The patient subsequently underwent seven cycles of chemotherapy with CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) and rituximab (monoclonal antibody to CD20 cell surface antigen). Treatment led to a dramatic reduction in the size of the mediastinal tumor, although the patient experienced no improvement in vocal fold function.

Patient 2. A 53-year-old man with a history of tobacco and alcohol abuse presented to the emergency department with a 2-week history of hoarseness and hemoptysis. He denied dysphagia, weight loss, fevers, sweats, and chills. Physical examination identified a 3-cm left thyroid nodule and rightward deviation of the cervical trachea. Chest x-ray detected multiple bilateral pulmonary nodules. Neck and chest CT demonstrated an enlargement of the left thyroid lobe with invasion of the adjacent larynx and trachea in addition to the pulmonary nodules (figure 2, A). The FNAC specimen contained monomorphic basaloid cells, hyaline globules, and cribriforming of the cells, all suggestive of adenoid cystic carcinoma (figure 2, B). Fiberoptic nasopharyngoscopy demonstrated fixation of the left vocal fold in the paramedian position and extramucosal compression of the left subglottis with no discrete mass or ulceration. In view of the somewhat conflicting clinical and cytopathologic findings, a transcervical incisional biopsy was performed. Histologically, the lesion displayed epithelial cells with the cylindromatous growth pattern typical of classic adenoid cystic carcinoma (figure 2, C), confirming the FNAC diagnosis.

The patient received 32 Gy of external-beam radiotherapy to the upper mediastinum and lower neck with palliative intent. He tolerated treatment well, his hemoptysis resolved, and his persistent dyspnea and hoarseness improved to the point that it was only mild. However, he died as a result of the pulmonary metastases 8 months after he had completed treatment.

Discussion

These two cases illustrate the fact that nonthyroid masses may mimic thyroid mass on examination and imaging, and that they can present with vocal fold paralysis. Although most paratracheal masses that cause vocal fold paralysis are of thyroid origin, physicians must be aware of the broad differential diagnosis of lesions that present in this manner. The differential diagnosis includes a variety of neoplastic, infectious, and inflammatory lesions of the lower neck and upper mediastinum (table). Approaching such lesions as thyroid neoplasms might unnecessarily expose patients to surgical risks (e.g., hypocalcemia, hypothyroidism, bilateral recurrent laryngeal nerve dysfunction, and airway compromise) without providing any benefit in terms of survival or disease control.

Figure 1. Patient 1. A: CT shows the extensive upper mediastinal mass and the deviation and compression of the trachea. B: The FNAC specimen contains spindle cells and a few lymphocytes (Diff-Quik stain, original magnification ×300). C: Histology demonstrates sheets of large atypical lymphoid cells with intervening fibrous tissue (H&E, original magnification ×300).
Even though the recurrent laryngeal nerves usually course deep to the thyroid, vocal fold paralysis is not common in patients with benign thyroid diseases or well-differentiated thyroid carcinoma. Anaplastic carcinoma of the thyroid, which is rare, has a propensity to involve adjacent structures, and it often presents with vocal fold paralysis. Therefore, among all patients with thyroid malignancies, the likelihood of vocal fold paralysis is low; however, patients with a thyroid mass who do have vocal fold paralysis have a high likelihood of malignancy.

Cytology. Given the broad differential diagnosis of paratracheal lesions that can cause vocal fold paralysis and their different treatment algorithms, pretreatment tissue diagnosis is of utmost importance. In most cases, a diagnosis can be obtained by FNAC. In fact, FNAC is widely considered to be the most cost-effective diagnostic test available for the evaluation of thyroid nodules. When performing cytology on a paratracheal mass, the first consideration is whether the lesion is of thyroid origin. This can usually be determined by the cellularity of the aspirate, the cell morphology, the presence or absence of colloid, and the colloid-to-cell ratio. With some lesions, this may not be quite so straightforward. For example, in patients with adenoid cystic carcinoma, such as our patient 2, a predominance of basaloid cells seen on FNAC may be mistaken for thyroid follicular cells, and hyaline globules may have the appearance of ropy colloid, suggestive of papillary carcinoma. If cell morphology proves to be unrevealing but sufficient aspirate is available, ancillary studies such as immunohistochemistry, flow cytometry, cytogenetics, and gene rearrangement often help classify the lesion. Immunohistochemical stains—including thyroglobulin, calcitonin, and thyroid transcription factor 1—can be used to determine thyroid origin. Markers such as cytokeratin and either CD3, CD20, or CD45 can be used to document carcinoma and lymphoma, respectively. In the event that FNAC findings are equivocal or the amount of aspirate is insufficient to allow for ancillary studies, tissue may be obtained by core needle or incisional biopsy. This will provide both cytologic and histologic clues, as well as sufficient tissue for ancillary studies.

Radiology. The two cases described herein underscore the importance of imaging in the workup of paratracheal masses. First, imaging may suggest the site of origin of the lesion. In our patient 2, the radiographic appearance was consistent with thyroid carcinoma invading the airway. Although this finding was actually misleading, in most cases of an aerodigestive tract malignancy that invades the thyroid or vice versa, the CT appearance will be suggestive. The case of our patient 1 illustrates how a mediastinal lesion may mimic a thyroid mass on physical examination but be readily apparent as such on CT. This finding narrowed the differential diagnosis during the evaluation of patient 1.
In addition, imaging is useful for investigating airway compromise. In patient 2, the airway compromise seen on CT led to the decision to perform an open biopsy under local anesthesia because endotracheal intubation might have caused airway edema and precipitated further compromise. Although imaging is seldom indicated in the workup of a thyroid nodule, the presence of vocal fold paralysis makes imaging essential. Thyroid carcinoma may cause vocal fold paralysis by placing pressure on or invading the recurrent laryngeal nerve, or it may cause vocal fold fixation by invading the larynx. Such an event may indicate the need for more extensive resection, possibly including the recurrent laryngeal nerve or a portion of the airway. Therefore, the information obtained by CT can be critical for patient counseling.

For the reasons we have outlined, we recommend a focused workup for all patients who present with a paratracheal lesion and vocal fold paralysis. This includes measurement of serum thyroid-stimulating hormone, which is commonly recommended in the cost-effective workup of any thyroid lesion. We also recommend CT of the neck down to the level of the aortic arch. Extending the neck CT to include the upper mediastinum or obtaining a separate chest CT is critical given that most unilateral vocal fold paralyses caused by malignancies are found in the chest. Ideally, FNAC should be performed following imaging because the resulted edema or hematoma formation may alter the radiographic appearance of the lesion and mislead the radiologist.