Recurrent Laryngeal Nerve Palsy on Integrated Positron Emission Tomography-Computed Tomography

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A 75-year-old woman with a long-standing history of smoking and emphysema presented with hoarseness and hemoptysis. Computed tomography (CT) scan found a large soft tissue mass involving the superior mediastinum and aortic-pulmonary window (Figure 1). Left main stem biopsy revealed small cell carcinoma. Apart from left vocal cord paralysis, no other laryngeal lesion was identified on bronchoscopy. Co-registered CT-Positron emission tomography (PET) fusion images identified physiologic fluorodeoxyglucose (FDG) uptake in the right vocal cord but no uptake in the left vocal cord (Figure 2).

Physiologic tracer uptake in the laryngeal muscles occurs during speech but not when a patient remains completely silent.1 When the recurrent laryngeal nerve is paralyzed, the ipsilateral vocal fold is fixed in a paramedian position due to the unopposed action of the cricothyroid muscle innervated by the superior laryngeal nerve.2 The compensatory movement during phonation of the contralateral vocal cord results in increased workload, and leads to increased local glucose consumption.

The combination of a left mediastinal mass with aorto-pulmonary window involvement or at the left lung apex (i.e., the anatomic course of the recurrent laryngeal nerve) and concomitant right-sided increased uptake in laryngeal muscles is a finding that can be seen in patients with recurrent left-sided laryngeal nerve palsy.3 Although FDG-PET is a sensitive imaging modality for lung cancer staging, anatomic localization of lesions detected at PET is suboptimal. Co-registered CT-PET fusion images allow the focal FDG uptake to be reliably localized to the small internal laryngeal muscles.

REFERENCES