An Unusual Cause of Stridor and Progressive Shortness of Breath*

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A 31-year-old man was referred to the pulmonary outpatient clinic for evaluation of difficult breathing of 1 year in duration. He was well until a year prior to consultation when noisy breathing developed, which was followed 6 months later by progressive shortness of breath. At the time of his evaluation, he was unable to walk a full city block. He had occasional cough productive of clear sputum, with nocturnal awakening to catch his breath. He had been previously worked up and treated for asthma with no improvement in symptoms while receiving bronchodilators and corticosteroids. He denied fever, orthopnea, paroxysmal nocturnal dyspnea, chest pain, weight loss, preceding upper respiratory infection, or recent travel.

The patient worked in an embroidery shop with exposure to lint for a few years without the use of a protective mask. Being at or away from work did not influence the symptoms of cough and dyspnea. He was a nonsmoker and had no history of childhood asthma. He underwent surgery 4 years earlier for a herniated disk under general anesthesia and was extubated postoperatively with no complications.

Physical examination revealed a young, healthy appearing man with stridorous breathing but in no acute distress. There was no sinus tenderness to palpation. The lung examination revealed no wheezing, and the cardiopulmonary examination was essentially normal. The rest of the head and neck examination was normal with no palpable neck masses or lymphadenopathy.

Pulmonary function test results showed the following: (1) spirometry: FVC before bronchodilator, 3.94 L (109% predicted); after bronchodilator, 4.13 L (114% predicted); FEV₁ before bronchodilator, 2.89 L (94.1% predicted); after bronchodilator, 2.98 L (96.8% predicted); FEV₁/FVC before bronchodilator, 73%; after bronchodilator, 72%; forced expiratory flow, midexpiratory phase, before bronchodilator, 2.41 L/s (68.6% predicted); after bronchodilator, 2.51 L/s (71.6% predicted); forced expiratory flow at 50% of vital capacity (VC)/forced inspiratory flow at 50% of VC (FIF₅₀) ratio before bronchodilator, 1.88; (2) lung volumes: total lung capacity, 5.25 L (103% predicted); VC, 4.10 L (114% predicted); residual volume, 1.15 L (77% predicted); functional residual capacity, 2.17 L (80% predicted); and (3) diffusion capacity: single-breath diffusion capacity of the lung for carbon monoxide, 27.3 mL/mm/mm Hg (88.8% predicted). Arterial blood gases on room air showed pH 7.4; P<sub>co₂</sub>, 41.5 mm Hg; P<sub>o₂</sub>, 106 mm Hg. The flow-volume loop (Fig 1) and chest radiograph of the patient (Fig 2) are shown.

What is your diagnosis?
Figure 1. Flow-volume loop.

Figure 2. Chest radiograph.
Diagnosis: Fixed upper airway obstruction secondary to infiltrating thyroid cancer

Upper airway obstruction can be a diagnostic challenge due to its relative rarity and because of its confusion with the more common causes of airflow obstruction, such as asthma and COPD. It is, however, an important cause of airflow limitation since it not only has the potential to produce acute airway compromise and respiratory failure but also may be amenable to curative surgery. The most common cause of upper airway obstruction in published series has been laryngeal and tracheal abnormalities. Other common causes include physical or chemical trauma to the major airways such as post-endotracheal tube stricture, posttracheostomy strictures, postthyroidectomy, bilateral cord paralysis with fixed stenosis, upper trachea strictures, tracheal tumors, thyroid goiters, vocal cord cancer, and idiopathic bilateral vocal cord paralysis with fixed stenosis.

Patients can present with a variety of complaints that may include dyspnea on exertion, stridor, wheezing, orthopnea, hemoptysis, hoarseness, and chest discomfort. Stridor is often misdiagnosed as wheezing related to asthma or vocal cord dysfunction. The best way to discriminate stridor from wheeze is based on timing and location. In contrast to wheezing, stridor is best heard on inspiration rather than expiration and is more prominent over the neck than over the chest. Vocal cord dysfunction, otherwise known as Munchausen stridor or factitious asthma, can also masquerade as asthma especially in women and may in fact coexist with asthma. Hence a careful history and physical examination are essential in establishing an early diagnosis.

Pulmonary function testing can aid in the diagnosis of upper airway obstruction. In the presence of large airway obstruction, the maximal flow-volume curve will demonstrate proportionate decreases in both the expiratory and inspiratory loops. A maximum expiratory flow at 50% of VC (maximum inspiratory flow 50% of VC ratio ≥ 1) and a FEV1 (milliliters)/peak expiratory flow rate (PEFR) [liters per min] of ≥ 10 mL/L/min are measurements that are found to be the most useful in distinguishing upper airway obstruction from normal and also from those with COPD. Other measurements such as FIF50 ≤ 100 L/min and FEV1/forced expiratory flow in 0.5 s ratio ≥ 1.5 could also distinguish upper airway obstruction from COPD and normal subjects but not as clearly.

The contour of the flow-volume loop can be used to further classify upper airway obstruction into three abnormal patterns. If the peak flows plateau on both the expiratory and inspiratory portion of the flow-volume loop and are decreased in approximately equal proportion, the lesion is classified as fixed. A marked reduction and plateau of inspiratory flow with a relatively normal expiratory flow-volume loop is classified as having variable extrathoracic obstruction. The third pattern, variable intrathoracic obstruction, demonstrates a reduction and plateau of the expiratory flow-volume loop with an intact inspiratory flow-volume loop.

Although a lot of information is known about symptomatic goiter as a cause of upper airway obstruction, there is paucity of information on the effects of infiltrating malignant thyroid masses on the flow-volume loop. The clinical course of papillary adenocarcinoma of the thyroid is delayed but aggressive. It is lethal in approximately 11 to 16% of patients, mostly due to respiratory obstruction resulting from extrinsic compression, intraluminal tumor extension, or unilateral or bilateral vocal cord paralysis. Dyspnea, dysphonia, dysphagia, hemoptysis, cough, sore throat, and a choking sensation are all signs and symptoms pointing to a tumor that is encroaching on the aerodigestive tract. Definitive treatment is total thyroidectomy, although removal of involved trachea and larynx may need to be done for more extensive disease. Tracheostomy is most useful in patients who are expected to have a limited airway postoperatively.

The present patient had a mild obstructive defect and no response to inhaled bronchodilator. The forced expiratory flow at 50% of VC (FIF50) ratio was 1.88, a finding that is consistent with upper airway obstruction. The flow-volume loop (Fig 1) also showed compromise of both inspiratory and expiratory loops. The constellation of findings on the pulmonary function test was characteristic of fixed upper airway obstruction. Interestingly, the chest radiograph also showed narrowing at the level of the trachea (Fig 2).

Urgent flexible fiberoptic laryngoscopy was performed, which revealed subglottic stenosis of approximately 3 mm in diameter with normal true vocal cord motion. A CT scan of the neck showed an infiltrating thyroid mass compressing the trachea at the level of the vocal cords with enlarged lymph nodes suspicious for metastatic disease (Fig 3).

The patient underwent total thyroidectomy and bilateral neck dissection with tracheoplasty after frozen section of the thyroid mass confirmed low-grade papillary carcinoma. The left internal jugular vein was also removed, as it was grossly involved with tumor. The final histopathology was low-grade papillary carcinoma with lymph node involvement.

Postoperatively, bilateral vocal cord paralysis was
noted for which a tracheostomy was performed. He did well postoperatively and was discharged home with a tracheostomy.

**Clinical Pearls**

1. Laryngotracheal obstruction is usually misdiagnosed as chronic obstructive lung disease or reactive airways disease. Hence, careful history taking and physical examination are important in establishing an early diagnosis.

2. Many of the lesions causing upper airway obstruction are amenable to curative surgery.

3. Goiters that cause upper airway obstruction are palpable, but infiltrating thyroid cancer as a cause may not be easily palpable.

4. Papillary adenocarcinoma of the thyroid is lethal in 11 to 16% of the patients, mostly due to respiratory obstruction resulting from extrinsic compression, intraluminal extension or spread, or unilateral or bilateral vocal cord paralysis.

5. A maximum expiratory flow at 50% of VC/maximum inspiratory flow at 50% of VC ratio ≥ 1 and a FEV₁/PEFR ratio ≥ 10 mL/L/min are useful measurements that can distinguish patients with upper airway obstruction from normal subjects and from those with COPD.

**Suggested Readings**


Rotman HH, Liss HP, Weg JG. Diagnosis of upper airway obstruction by pulmonary function testing. Chest 1975; 68: 796–799