Iatrogenic Tracheal Stenosis Presenting as Persistent Asthma

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Although the incidence of post-intubation tracheal stenosis has markedly decreased with the advent of large volume, low pressure endotracheal tube cuffs, it still occurs, commonly in patients after prolonged intubation. We report a case of tracheal stenosis that developed after a brief period of endotracheal intubation, and that was misdiagnosed and treated as asthma and panic attacks. *Key words: asthma; tracheal stenosis; wheezing; airway disease; interventional bronchoscopy*. [Respir Care 2013;58(9):e107–e110. © 2013 Daedalus Enterprises]

Introduction

Tracheal stenosis is a common complication of tracheostomy, as well as endotracheal intubation. In recent years the incidence of post-intubation tracheal stenosis has markedly decreased with the advent of large volume, low pressure endotracheal tube cuffs, but tracheal stenosis still affects patients requiring prolonged intubation.[1] We report a case of tracheal stenosis that developed after a brief period of endotracheal intubation, and that was misdiagnosed and treated as asthma and panic attacks.

Case Report

A 30-year-old, lifelong non-smoker was evaluated in the emergency room after his fourth visit to the hospital for symptoms of dyspnea, wheezing, and chest tightness. His medical history was notable for panic attacks and intubation for 48 hours after a bout of pneumonia 8 months earlier. He denied a history of asthma, but reported environmental, feline, pollen, and cephalosporin allergies. His symptoms included progressively worsening exertional dyspnea over the past month. He also reported heart palpitations and episodic non-productive cough. At each previous emergency room visit he was given albuterol and discharged on oral prednisone without symptom relief. He was single and unemployed.

On physical examination his temperature was 37°C, heart rate 88 beats/min, blood pressure 122/74 mm Hg, breathing frequency 18 breaths/min, and S_{pO_2} 98%. Auscultation revealed mild bilateral expiratory wheezes and audible breathing from his mouth. Chest radiograph was normal. Computed tomography (CT) angiogram was negative for pulmonary embolism. FVC was 5.12 L (88% of predicted), FEV_1 was 2.22 L (47% of predicted), and FEV_1/FVC was 43%. Static lung volumes were normal. Figure 1 shows his flow-volume loop.

Cervical CT revealed stenosis involving the upper third of the trachea (Fig. 2). Bronchoscopy revealed normal vocal cords. Two centimeters below the vocal cords there was a circumferential stenosis producing 85% obstruction (Fig. 3). Initiation of dexamethasone and bronchodilators produced minimal symptom improvement. He was referred to a tertiary center for surgery.

Discussion

Tracheal stenosis is a rare but serious complication of endotracheal intubation. The incidence declined following the introduction of high-volume, low-pressure endotracheal tube cuffs. The reported incidence has varied. In one prospective study, 11% of patients intubated with high-volume, low-pressure cuffed tubes developed tracheal stenosis, exhibited by 50–90% narrowing of the tracheal lumen at the cuff site.[2] Another study reported an incidence of 4.9

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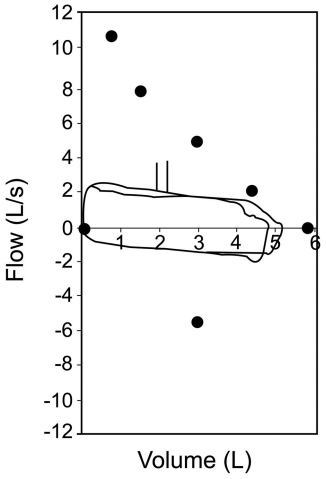

Fig. 1. Flow-volume loop revealing fixed upper-airway obstruction.

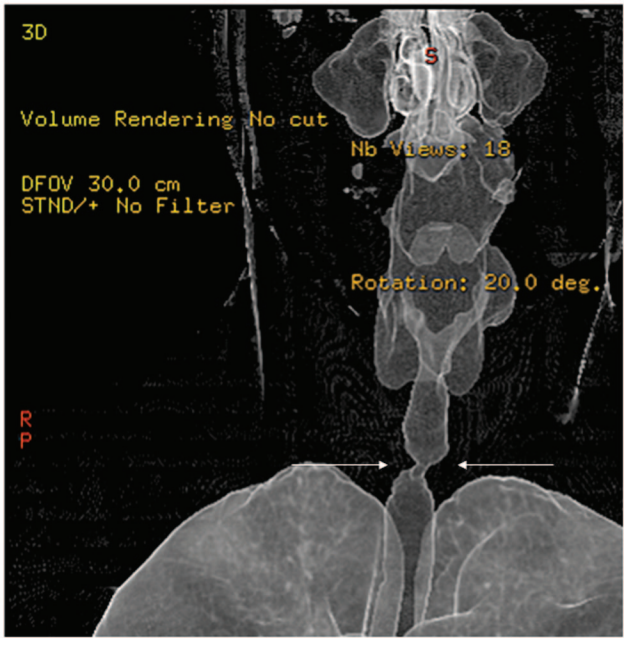

Fig. 2. Three-dimensional computed tomogram shows stenosis of the upper trachea (arrows).

cases per million per year in a general population.[3] The overall incidence of post-intubation and post-tracheotomy stenosis ranges from 6% to 22%, but only 1–2% of the patients are symptomatic or have severe stenosis.[1,4]

The etiology of post-intubation tracheal stenosis is multifactorial. One of the most important causes is cuff pressure that exceeds the mucosal capillary perfusion pressure (20–30 mm Hg). High cuff pressure causes ischemic injury as rapidly as 15 min after intubation. This ischemia leads to ulceration, chondritis, and, eventually, fibrosis within 3–6 weeks.

Another factor is the duration of intubation. The onset of stenosis usually ranges from 2–24 weeks following extubation,[5] but the incidence increases with the duration of intubation, and tracheal stenosis is rare when intubation was < 1 week.[6] Whited found a 12% incidence of laryngeal stenosis in patients who were intubated for ≥ 11 days, a 5% incidence following 6–10 days of intubation, and a 2% incidence with < 6 days of intubation.[7] To our knowledge, only 3 cases of post-intubation tracheal stenosis have been reported with intubation of < 48 hours. One patient developed tracheal stenosis after 10–12 hours of intubation, and the 2 other cases manifested at 24 and 48 hours.[8,9] Our patient developed post-intubation tracheal stenosis after only 48 hours of intubation, making it extremely rare.

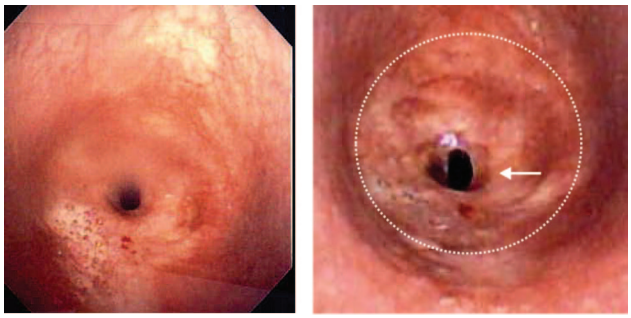

Fig. 3. Left: Bronchoscopy shows circumferential stenosis. Right: The dotted line shows the normal diameter of the trachea. The arrow points to the stenotic ring.

Other factors include endotracheal tube size, hypotension at the time of intubation, high tracheotomy site, traumatic events during intubation, steroid use, advanced age, severe reflux disease, autoimmune diseases, obstructive sleep apnea, and radiation therapy.[1,10]

Most patients who undergo endotracheal intubation develop some degree of stenosis.[11] However, patients usually remain asymptomatic until stenosis narrows the trachea to approximately 70% of its original lumen.[10] In patients with a lesser degree of stenosis, diagnosis is more challenging. These patients may present with recurrent pneumonia or progressive dyspnea on exertion.[12] Such patients are often misdiagnosed and treated for asthma or chronic bronchitis, as in our patient.[10] Stridor is not usually seen until the tracheal lumen is < 5 mm, and cyanosis may be a late finding.[5] The differential diagnosis for tracheal stenosis is usually categorized as focal or diffuse (Table).

Diagnosis is aided by spirometry, bronchoscopy, and reconstructive imaging. The flow-volume loop typically reveals a fixed obstructive pattern, with flattening of the inspiratory and expiratory curves (see Fig. 1).[13] Bronchoscopy is the diagnostic modality of choice, as it can exclude diseases with similar presentations, such as tracheomalacia and vocal cord paralysis, while at the same time providing therapeutic modalities. While imaging techniques have become faster and allow for anatomic detail, conventional radiography is often the first step in the evaluation of suspected central airway disease, and may be adequate to

Table. Differential Diagnosis of Tracheal Diseases

Focal Disease
Post-intubation stenosis
Post-infectious stenosis
Post-transplant stenosis
Systemic disease
Crohn disease
Sarcoidosis
Behçet disease
Diffuse disease
Wegener granulomatosis
Relapsing polychondritis
Tracheobronchopathia osteochondroplastica
Amyloidosis
Papillomatosis
Rhinoscleroma

identify abnormalities. However, CT improves both the detection and characterization of central airway disease entities and may show findings such as calcifications that aid in the planning of bronchoscopy or therapy. Furthermore, the 3-dimensional CT we used in our patient is a powerful tool that facilitates therapy selection.

The treatment for tracheal stenosis has changed dramatically over the years[14] and requires a comprehensive multidisciplinary approach. The primary objective of airway reconstruction in post-intubation tracheal stenosis is to restore a patent airway while minimizing the risk of further iatrogenic injury. Tracheal resection followed by end-to-end anastomosis is the standard of care for tracheal stenosis, and has a success rate of up to 97%.[15,16]

Advances in endoscopic interventions have improved the treatments options for patients with tracheal stenosis.[17] Minimally invasive treatment, including neodymium-yttrium-aluminum-garnet laser (Nd:YAG) and CO_2 laser excision, dilatation, endoscopic stent placement, and topical application of mitomycin C, reduce airway surgical manipulation and post-surgical inflammatory response.[18] Still, for better outcome, endoscopic management requires early referral for stenotic lesions shorter than 10 mm, or shorter than 30 mm if stenting is considered, with no circumferential scarring and no loss of cartilagenous support.[15,16] Balloon dilatation has been well tolerated, but was often followed by recurrence requiring repeated dilatation or surgery. Improvements and advances in endoscopic techniques and topical mitomycin C have expanded treatment options for tracheal stenosis with excellent outcomes.[18]

While some argue that rigid bronchoscopy remains the therapeutic standard, it is now used less frequently. While tracheal resection and reconstruction have predictable, reliable results, airway stents are now used for both benign and malignant central airway obstruction. Stents fall into 2 major categories: silicone and self-expanding metallic.[19] Stent selection for tracheal stenosis is based on many factors, including operator comfort. Complications are associated with all types of stents. Smaller studies have reported long-term silicone stenting to be effective in curing post-intubation stenosis in 50% of patients, with removal of the stent at 1 year. Less than half the patients required any follow-up interventions. Airway obstruction from tracheal stenosis usually involves a very short segment of the trachea and can be circumferential, as in our case, or anterior, depending on etiology (see the Table).

Tracheal stenosis is a serious complication following intubation. In patients with a history of intubation who present with respiratory symptoms, tracheal stenosis should be included in the differential diagnosis.

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