American Journal of Hospital Medicine
Promoting research and education in the field of hospital medicine. ISSN 2474-7017 (online)

Case Reports

Symptomatic Tracheal Stenosis Masking As COPD Exacerbation: A Case Report

January 15, 2016 Case Reports, January-March 2016: Volume 8 Issue 1
Keywords Tracheal stenosis

Symptomatic Tracheal Stenosis Masking As COPD Exacerbation: A Case Report

Lauren E. Smith, MS-IV[1]*, Prasad S. Garimella, M.D.[2]

Institutions:

[1]Philadelphia College of Osteopathic Medicine – GA Campus, 625 Old Peachtree Road NW, Suwanee, GA 30024

[2]631 Professional Drive, Suite 350, Lawrenceville, GA 30046

Email: [1]LaurenSm@pcom.edu; [2]psgarimella@gmail.com

* = Corresponding Author

Abstract:

Tracheal stenosis can result from multiple conditions such as traumatic injury, benign and malignant lesions, inflammatory illnesses, and congenital diseases. Despite technological advancement, the leading cause continues to be injury from endotracheal intubation and tracheostomy. Tracheal stenosis occurs in approximately 6-21% of patients with intubation and/or tracheostomy, while only 1-2% of this population actually becomes symptomatic. The usual manifestations are exertional dyspnea, wheezing, and stridor which can often be mistaken for an asthma or COPD exacerbation. In this article, we describe a 72-year-old Caucasian female with a history of COPD who developed symptomatic tracheal stenosis following prolonged intubation and tracheostomy that was initially misdiagnosed and treated as a COPD exacerbation.

Keywords: Endotracheal Intubation; Tracheostomy; Complication; COPD

Introduction:

For over a century, tracheal stenosis has been regarded as a cause of upper airway dysfunction. The first recognized cases of tracheal stenosis date back to 1886 following tracheotomy for croup and diphtheria.[1]The etiology of tracheal stenosis is multifactorial; however, despite technological advancement, the leading cause continues to be endotracheal intubation and tracheostomy. In this article, we report the case of a 72-year-old female with a history of COPD who developed symptomatic tracheal stenosis following prolonged intubation and tracheostomy that was initially misdiagnosed and treated as a COPD exacerbation.

Report of the Case:

A 72-year-old Caucasian female with a history of COPD presented to the emergency room complaining of progressive shortness of breath over the past few days. The patient was a former tobacco user with a history of 50 pack years of smoking. Seven months prior, she had been hospitalized following trauma from a MVA and she was mechanically ventilated with an endotracheal tube for 36 days. She suffered prolonged respiratory failure to wean off mechanical ventilation and H. influenzae pneumonia during her hospital stay. The patient was eventually transferred to a long-term acute care facility to continue rehabilitation and ventilatory weaning where she subsequently underwent a tracheostomy for long-term care.

Two months following this transfer, she returned from the LTAC facility due to wound breakdown and hardware exposure at a previous traumatic injury site. She underwent surgical debridement and hardware removal. During this hospitalization, her respiratory status was deemed stable to allow for decannulation of the tracheostomy. The patient was discharged home.

The current emergency room visit is five months following the decannulation of the tracheostomy. In addition to COPD, her past medical history was notable for hypothyroidism, hypertension, hyperlipidemia, atrial fibrillation, diastolic congestive heart failure, and coronary artery disease with previous CABG. Besides the progressive shortness of breath, the patient also complained of occasional cough with no sputum production. She denied chest pain, palpitations, fever, chills, nausea, and vomiting. The remainder of the review of systems was non-contributory.

Upon physical examination, the patient appeared anxious and was in moderate respiratory distress with a tripod positioning on the stretcher. Vital signs included a temperature of 97.9 degrees Fahrenheit, blood pressure of 165/94 mm Hg, heart rate of 120, respiratory rate of 32, and oxygen saturation of 87 percent. Cardiac auscultation demonstrated distinct S_1 and S_2 heart sounds with an irregular rhythm and tachycardia. No gallops, murmurs, or rubs were audible. The patient exhibited equal chest expansion; bibasilar rales were audible throughout the lungs. The neck was supple with no evidence of JVD or tracheal deviation; however, mild inspiratory and expiratory sounds over the trachea were noted. The rest of the examination was unremarkable.

Initial laboratory data revealed a mildly elevated WBC at 12.9; hemoglobin, hematocrit, and chemistries were within normal ranges. An arterial blood gas demonstrated evidence of respiratory acidosis with a pH of 7.32 and PCO_2 of 60 mm Hg, and a compensated HCO_3 of 30.

The patient was placed on BiPAP and admitted to the hospital for further treatment regarding suspected congestive heart failure and COPD exacerbation. She was placed on a diuretic, and she was given Symbicort and albuterol nebulizer treatments to improve her lung function.

The chest radiograph showed cardiomegaly and mild bibasilar atelectasis; no pneumonia or evidence of pulmonary edema was noted. An echocardiogram was performed and demonstrated a normal-sized left ventricle with an ejection fraction of 45%. Tricuspid regurgitation was again noted as the cause for her diastolic heart dysfunction.

Despite treatment, the patient continued to experience progressive respiratory distress. Due to the previous history of long-term intubation and tracheostomy and persistence of the patient's symptoms, tracheal stenosis was considered. A CT of the neck was performed and revealed moderate focal subglottic tracheal stenosis 2.46 cm below the vocal cords (Figure 1) with a transverse luminal diameter of 4.52 mm (Figure 2). The AP luminal diameter was 11.6 mm at the level of stenosis (Figure 3). The transverse luminal diameters above and below the area of stenosis were 1.4 cm and 1.5 cm, respectively.

The patient was transferred to a nearby academic center for further treatment with rigid bronchoscopy dilatation and possible endotracheal stent placement.

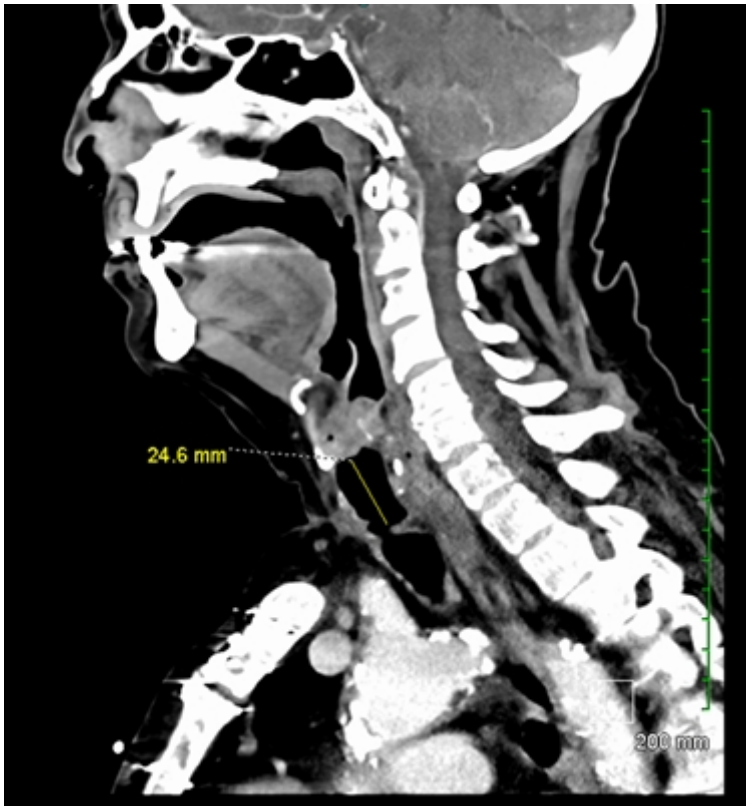

Figure 1. Length of Stenosis below Vocal Cords. CT scan of the neck revealed moderate focal subglottic tracheal stenosis 2.46 cm below the vocal cords.

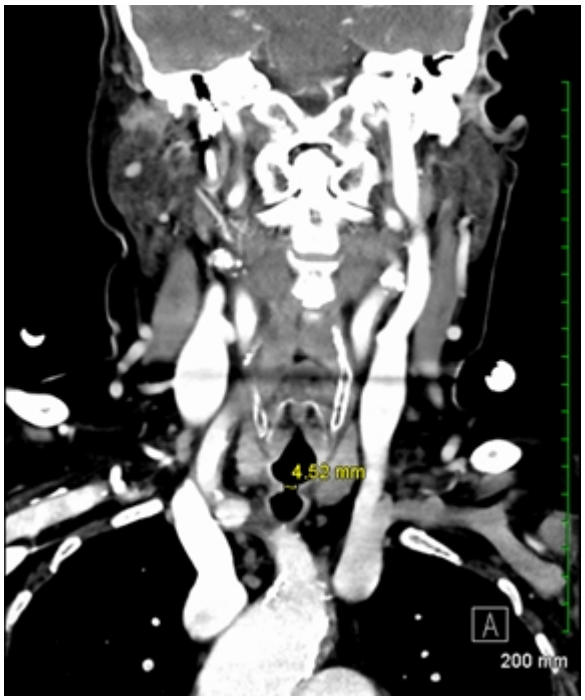

Figure 2. Transverse Diameter at the Level of Stenosis. There is a tracheal transverse luminal diameter of 4.52 mm at the level of stenosis.

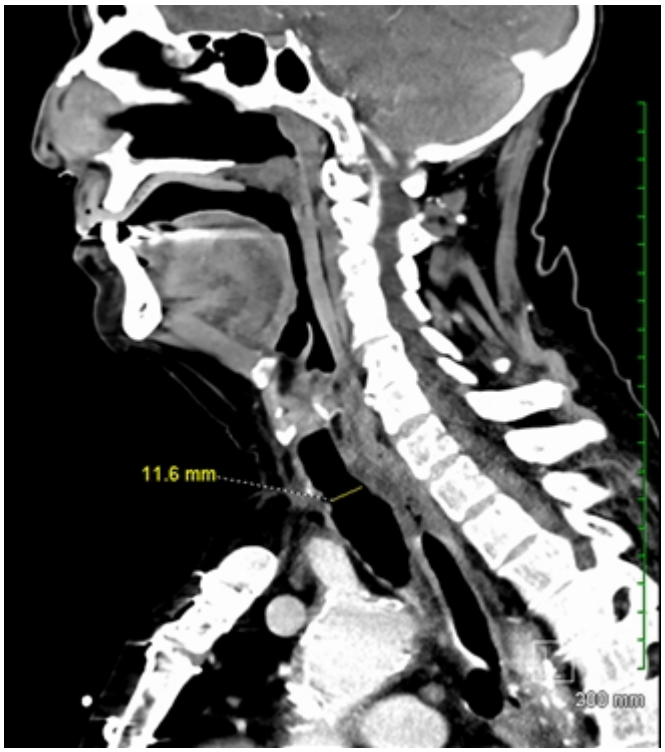

Figure 3. AP Diameter at the Level of Stenosis. There is a tracheal AP luminal diameter of 11.6 mm at the level of stenosis.

Discussion:

Tracheal stenosis can result from multiple conditions such as traumatic injury, benign and malignant lesions, inflammatory illnesses, such as Sarcoidosis and scleroderma, and collagen vascular diseases, such as Wegner's granulomatosis. Despite technological advancement, the leading cause continues to be injury from endotracheal intubation and tracheostomy. Endotracheal intubation and tracheostomy are the most frequently performed procedures on critically ill patients. Late complications of these procedures, such as tracheal stenosis, are largely underreported because many patients may die before decannulation and proper follow-up. Recent studies suggest a lower incidence of tracheal stenosis occurring in approximately 6-21% of patients with intubation and/or tracheostomy, while only 1-2% of this population actually becomes symptomatic.[2,3,4] However, other studies have suggested higher rates of incidence up to 65%.[5]

The most common sites of stenosis arethe site of endotracheal tube cuff and the tracheostomy stoma site. Stenosis due to endotracheal intubation usually results in a web-like stenosis. The pathogenesis is believed to originate from the loss of mucosal blood flow secondary to cuff pressure greater than 30 mm Hg on the tracheal wall. Ischemia occurs within hours thus leading to the development of necrosis, destruction of the tracheal architecture, and the formation of fibrotic tissue. Stenosis due to tracheostomy is often more complex and it is believed to result from abnormal wound healing with excess granulation tissue and circumferential scar contraction. [4,5,6,7,8,9]

Risk factors that encourage the development of tracheal stenosis include: diabetes mellitus, hypertension, cardiovascular disease, current tobacco use, female gender, autoimmune dysfunction, history of radiation therapy, and steroid use. It is hypothesized that many of these comorbidities contribute to microvascular occlusion and mucosal ischemia. In addition, a history of radiation therapy and steroid use may also alter the healing process within the trachea leading to increased fibrosis and tracheomalacia. Numerous studies have found tracheal stenosis to more commonly occur in females.[6,10,11,12] The reason for this has remained largely controversial. It has been thought that estrogen increases the levels of TGF β1 thus promoting increased collagen deposition and fibrosis.[5,13] The likelihood of tracheal stenosis development is also largely linked to the length of intubation. A study conducted by Whited[14] found that patients who were intubated greater than 11 days had a 12% risk of developing tracheal stenosis; whereas, patients intubated 6-10 days had a 5% risk and those intubated less than 6 days had a 2% risk, respectively.

Tracheal stenosis is largely asymptomatic until the stenosis becomes greater than thirty percent of the original tracheal diameter.[9,15] The first sign of airway obstruction due to tracheal stenosis presents as exertional dyspnea. Wheezing or stridor occurs once the tracheal lumen is less than five millimeters.[8,9,10] Flow volume curves may demonstrate evidence of obstruction but are of little practical diagnostic use due to severity of respiratory dysfunction and underlying lung disease, such as COPD and asthma. In fact, numerous case reports recount patients with tracheal stenosis who were initially misdiagnosed as experiencing an asthma exacerbation.[9,10,11] A chest x-ray is ineffective because the lungs are typically clear radiographically. Linear x-ray studies of the neck and CT imaging are considered the imaging modalities of choice. Direct bronchoscopic examination offers a definitive diagnosis.[8]

There are two widely accepted classification systems for tracheal stenosis: the Myer-Cotton system and the McCaffrey system. The Myer-Cotton system primarily addresses circumferential stenosis confined to the subglottic region; it grades the level of stenosis based on the relative reduction in cross-sectional area of the trachea. Grade I lesions have less than 50% obstruction, grade II lesions have 51-70% obstruction, grade III lesions have 71-99% obstruction, and grade IV lesions have complete stenosis.[16] On the other hand, the McCaffrey system classifies tracheal stenosis based on the length and subsites involved. Stage I lesions are confined to the subglottis or trachea and are less than 1 cm long, stage II lesions are isolated to the subglottis and are greater than 1 cm in length, stage III are subglottic/tracheal lesions not involving the glottis, and stage IV lesions involve the glottis.[17]

The treatment for tracheal stenosis has evolved over time. The current treatment modalities include: endoscopic therapies with laser photo-dissection and dilation, stenting with silicone or metal stents, and surgical repair with primary resection and anastomosis or multistage laryngotracheoplasty. Brichet, et al.[15] designed a treatment algorithm utilizing a multidisciplinary approach to tracheal stenosis management. The use of laser photo-dissection and dilatation for less severe, web-like stenosis was recommended. On the other hand, stent implantation was advised for more complex stenosis containing extensive scar formation and tracheomalacia. After six months of stent placement, patients were evaluated for surgical repair. Those who were surgical candidates underwent surgical resection and reconstruction with success rates up to 97%. Patients who were not deemed fit for surgery maintained their tracheal stents.[8,9,10]

Conclusion:

There is a quantifiable risk for tracheal stenosis complication following endotracheal intubation and/or tracheostomy. As a result, physicians should have a high clinical suspicion in patients with a history of these procedures who present with progressive dyspnea and wheezing found to be unresponsive to medical therapy for asthma or COPD.[3,9] In addition, it is advised that patients who underwent endotracheal intubation or tracheostomy procedures obtain routine endoscopic or radiologic tracheal assessment within a few months following de-cannulation to monitor the development of stenosis.[5,12]

Abbreviations:

COPD: Chronic Obstructive Pulmonary Disease; MVA: Motor Vehicle Accident; LTAC: Long-Term Acute Care; CABG: Coronary Artery Bypass Graft; JVD: Jugular Venous Distension

Acknowledgements:

The authors would like to thank all of the healthcare providers who participated in this patient's care.

References:

[1]Colles CJ. On stenosis of the trachea after tracheotomy for croup and diphtheria. . 1886;3:499-507.

[2]Grillo HC, Donahue DM, Mathisen DJ, Wain JC, Wright CD. Postintubation tracheal stenosis. Treatment and results. . 1995;109:486-493.

[3]Norwood S, Vallina VL, Short K, Saigusa M, Fernandez LG, McLarty JW. Incidence of Tracheal Stenosis and Other Late Complications After Percutaneous Tracheostomy. . 2000;232(2):233-241.

[4]Tzouvelekis A, Kouliatsis G, Oikonomou A, et al. Post-intubation pulmonary embolism and tracheal stenosis: A case report and review of the literature. . 2008;102(8):1208-1212.

[5]Stauffer JL, Olson DE, Petty TL. Complications and Consequences of Endotracheal Intubation and Tracheotomy. A Prospective Study of 150 Critically Ill Adult Patients. . 1981;70(1):65-76.

[6]Zias N, Chroneou, A, Tabba MK, et al. Post Tracheostomy and Post Intubation Tracheal Stenosis: Report of 31 Cases and Review of the Literature. . 2008;8:18-26.

[7]Esteller-Moré E, Ibañez J, Matiño E, Ademà JM, Nolla M, Quer IM. Prognostic factors in laryngotracheal injury following intubation and/or tracheotomy in ICU patients. . 2005;262:880-883.

[8]Grillo HC, Donahue DM. Postintubation tracheal stenosis. . 1996;6(4):725-731.

[9]Spittle N, McClusky A. Lesson of the week: Tracheal stenosis after intubation. . 2000;321:1000-1002.

[10]Barreiro TJ, Ghattas C, Valino CA. Iatrogenic Tracheal Stenosis Presenting as Persistent Asthma. . 2013;58(9):e107-110.

[11]Zubairi AB, Dildar B, Husain SJ, Khan MF. Tracheal stenosis mimicking severe acute asthma. . 2010;10.1136.

[12]Dane TE, King EG. A Prospective Study of Complications after Tracheostomy for Assisted Ventilation. . 1975;67(4):398-404.

[13]Nesek-Adam V, Mršić V, Oberhofer D, Grizelj-Stojčić E, Košuta D, Rašić Ž. Post-intubation long-segment tracheal stenosis of the posterior wall: a case report and review of the literature. . 2010;24:621-625.

[14]Whited RE. Laryngeal dysfunction following prolonged intubation. . 1979;88:474-478.

[15]Brichet A, Verkindre C, Dupont J, Carlier ML, Darras J, Wurtz A. Multidisciplinary approach to management of postintubation tracheal stenosis. . 1999;13:888-893.

[16]Cotton RT, Richardson MA. Congenital laryngeal abnormalities. . 1981;14:203-218.

[17]McCaffrey TV. Classification of laryngotracheal stenosis. . 1992;102:1335–1340.