Postintubation Tracheal Stenosis: Case Report and Review of Current Management

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Abstract Tracheal stenosis is an uncommon but serious complication of prolonged intubation and tracheostomy patients. Treatment is often provided by otorhinolaryngologists or interventional pulmonologists, but because symptoms may not appear for weeks or months after extubation, primary care physicians are frequently the first to encounter this complication. On presentation, tracheal stenosis may be mistaken for asthma with dyspnea and stridor. Presented is the case of a 53-year-old woman who was referred to the otorhinolaryngologist with post-intubation tracheal stenosis and underwent a series of surgical procedures to repair her airway including tracheal resection and anastomosis. The common presenting signs, treatment options, and prevention strategies for similar patients are examined.

Keywords tracheal stenosis; stridor; dyspnea; tracheal resection

1. Introduction

Tracheal stenosis has various etiologies, including trauma from surgery, intubation, or accidents, inhalation injuries, or inflammatory diseases such as Wegener’s granulomatosis, sarcoidosis, or systemic lupus erythematosus [7]. Currently, however, the vast majority of cases are the result of prolonged intubation or tracheostomy. Even when high volume, low pressure cuffed tubes are used, airway stenosis may occur in up to 11% of intubated or tracheostomy patients [10], even after less than 24 hours of intubation [2, 3]. When tracheal stenosis occurs, it is often difficult to manage and definitively treat. The common presentation of tracheal stenosis is with general respiratory symptoms of wheezing, dyspnea, exercise intolerance, stridor, cough, or especially in children, an otherwise mild respiratory infection leading to acute respiratory distress [4]. These symptoms may not initially suggest an airway stenosis and may be misdiagnosed as asthma, a far more common condition, especially in the pediatric population. However, if a dyspneic patient has a history of intubation or tracheostomy, tracheal stenosis should be considered. By the time of symptomatic presentation, the stenosis has usually progressed to involve 30–50% of the lumen diameter. The Myer-Cotton system for grading of stenoses is seen in Table 1. By 80% occlusion, the patient will be in acute distress [4, 9]. This diagnosis is missed in the initial presentation in up to 44% of these patients. Delay in proper treatment leads many patients to require emergency bronchoscopic dilation because the stenosis has progressed so severely before the problem is correctly identified [1, 2].

2. Case report

A 53-year-old Caucasian female was referred to the otorhinolaryngology clinic with a history of prolonged intubation a year previously, following a suicide attempt. She was tracheostomy-dependent and a noncontrast CT scan revealed 11 mm of occluded trachea superior to the tracheostomy tube. An initial rigid bronchoscopy dilated the pinpoint tracheal lumen, first with a urethral filiform catheter until the lumen was opened enough for use of an angioplasty catheter, then rigid Jackson dilators. The procedure also involved excising significant stomal granulation tissue, and placing a T-tube to maintain the patent airway. Over the next year, the patient experienced repeated infection of the T-tube and underwent multiple surgeries for replacement. When this resulted in no definitive solution, a tracheal resection and primary anastomosis was performed. She was extubated a week later and had no complications with eighteen months of follow-up and no evidence of restenosis.

3. Discussion

Tracheal stenosis is most commonly acquired from prolonged intubations in which the endotracheal cuff pressure...
exceeded the mean capillary pressure of the tracheal mucosa (> 30 mmHg). The excessive pressure leads to ischemia, granulation tissue formation, and scarring with lumen stricture [2,4,5]. The result is a web-like fibrosis, usually with minimal damage to the cartilaginous support structure.

A second common cause of tracheal stenosis is via tracheostomy damage. The injury may involve fractured cartilage from mechanical leverage of the ventilator tubing on the tracheal tube, incorrect sizing of the tracheostomy, fracture during percutaneous tracheostomy tube placement, and excess granulation tissue from infection and abnormal healing [8,11].

Patients may begin the process of developing tracheal stenosis even if intubated less than 24 hours [2,3]. However, with intubations of any length, symptoms may not present for several months after extubation [10]. This characteristic increases the likelihood that the patient will present first to a primary care physician with dyspnea.

The development of high-volume, low-pressure intubation cuffs has greatly reduced the incidence of postintubation stenosis, but it is still as high as 11% among patients with a history of prolonged intubation [10]. The incidence could be further reduced with careful endotracheal tube and stoma care, including regular documentation of the cuff pressure and support for the ventilator equipment.

Common comorbidities of tracheal stenosis include cardiovascular disease, diabetes mellitus, and conditions requiring chronic corticosteroid use, all of which increase the microvascular damage and impair the healing process [11].

Rigid bronchoscopy is regarded as the most definitive diagnostic tool for observing the location of the lesion, diameter of the airway lumen, and length of stenosis. Computed tomography also provides an image of the narrowing, especially if lumen is too narrow to allow for a bronchoscope to pass through [2,4].

For many years, open surgery with resection and anastomosis was regarded as the gold standard of treatment for nearly every tracheal stenosis patient, and the only way to avoid recurring problems. Several recent studies, however, have pointed out that a significant portion of postintubation tracheal stenosis patients are unable to undergo open surgery due to significant comorbid conditions. For these cases, endoscopic procedures have improved to become viable treatment options for select patients [1,2,5,6,9]. The ideal treatment plan must be individualized and balanced between a more definitive but invasive surgical resection with end-to-end anastomosis or the more conservative endoscopic treatments, which have greater risk of recurrence. Useful algorithms for treatment selection are now available, using features of the case including length of the stenosis, lumen diameter, cartilage involvement, and prior treatment attempts [1,5].

Endoscopic procedures currently used include dilation, stenting, excision of granulation tissue by electrocautery, laser, or sharp incision, and topical application of steroids or mitomycin C. These treatments are the primary choice for elderly or very ill patients for whom open surgery would be difficult. Additionally, stenotic lesions < 20 mm in length, without cartilage damage, may be more easily treated endoscopically, without requiring resection. This practice was demonstrated on the 11 mm lesion in the case presented. Balloon dilation or stent placement is usually the initial endoscopic treatment; it may be combined with either CO₂ or neodymium-yttrium aluminum garnet (Nd-YAG) laser excision of granulation tissue and topical application of mitomycin C (MMC). MMC reduces granulation through inhibition of fibroblasts and DNA synthesis [10]. Argon plasma coagulation (APC) has also been used to control bleeding prior to excision of granulation tissue [10].

Balloon dilations are often repeated, perhaps 3 times or more before a cure is reached. If restenosis occurs a fourth time, resection or stent placement is often reconsidered for more definitive treatment [1]. The number of repeated dilation attempts should be determined by successful progression of lumen widening with previous dilations [6]. The patient presented required a dilation procedure initially, then several operations to replace T-tubes due to infection and granulation tissue.

Stents have more complications including migration, obstruction, and granulation formation [1,6]. However, Brichet et al. [1] observed that if a stent was left in place for several months, the stenosis could mature and stiffen, resolving the problem even when the stent was removed.

In cases involving > 30 mm of stenosis, cricoid damage, or loss of cartilaginous support, successful treatment outcomes are greatly increased with open surgical resection and primary anastomosis over endoscopic management [5,6]. Nouraei et al. [6] studied 62 patients with postintubation tracheal stenosis treated with only endoscopic procedures; success rates fell from 96% to 20% if the lesions were longer than 30 mm. Also, patients who have had repeated complications after endoscopic treatment attempts may have more definitive outcomes with open surgical resection and anastomosis, such as in the case presented.

Restenosis is usually within 1 to 3 months after the surgical resection and anastomosis when it occurs [10]. Mortality rate for this operation is about 3% [10] and failure rate (requiring tracheostomy tube replacement) is about 15% [1,2]. Most common complications include cervicomedial sepsis, dehiscence of the sutures, rupture of the innominate artery, aspiration pneumonia, and palsy of the recurrent laryngeal nerve [1]. Grillo et al. [3] determined that using absorbable sutures nearly eliminated the risk of granulations along the anastomosis, which had been a very common cause of restenosis.
In summary, patients with a history of prolonged intubation or tracheostomy who present with stridor, dyspnea, cough, or wheezing should be evaluated for postintubation tracheal stenosis. In cases with stenoses < 20 mm and intact cartilage, or those involving patients who are not good candidates for surgery, endotracheal dilation or stenting with possible application of mitomycin C may be a valuable treatment option. Patients with longer and more complex stenoses (loss of cartilage support or repeated complications) will likely require resection and end-to-end anastomosis for definitive treatment.

References