A Tracheostomy Complication Resulting from Acquired Tracheomalacia: A Case report

Bach T. Le, MD, DDS, James M. Eyre, Jr., MD, DMD, Eric P. Holmgren, MS, Eric J. Dierks, MD, DMD, FACS

Introduction

Intraoperative complications of tracheostomy are well described in the literature. These include hemorrhage, perforation of the walls of the trachea and esophagus, recurrent laryngeal nerve injury, intraoperative fire, pneumothorax and pneumomediastinum, and tube displacement. We present an unusual complication encountered during a routine tracheostomy that resulted from severe tracheomalacia.

Case Report

A 65-year-old Caucasian female was found at home unresponsive in pulseless electrical activity. She was intubated and resuscitated in the field, transported to the hospital, and then admitted to the intensive care unit (ICU) by the medicine service. The resuscitation continued upon arrival to the hospital. She remained intubated with an ongoing problematic air leak around her cuff. To adequately ventilate the patient, the endotracheal tube (ETT) cuff required increasing volumes (pressures) to provide an adequate seal. Our service was consulted 10 days after admission to perform a tracheostomy because of anticipated prolonged ventilator dependency.

The patient's past medical history included a long-standing history of chronic obstructive pulmonary disease (COPD) and asthma with a history of prior ICU admissions and intubations, hypertension, hypertrophic cardiomyopathy with ventricular arrhythmias, cerebral vascular accidents and seizures. The patient also had a long history of smoking.

On hospital day 10 the patient was taken to the operating room for the planned tracheostomy procedure. Upon uncomplicated placement of a # 8 Portex tracheostomy tube, under direct vision into the tracheal lumen, a large leak around the cuff was immediately noted. It was decided to replace it with a #9 Portex tracheal tube. The tracheostomy tube exchange occurred under direct visualization. In the process of the exchange, the patient became progressively difficult to ventilate with increasing airway pressures. Within minutes, the patient went through an acute desaturation phase down to 30%.   The patient was treated for a presumed bronchospasm and asthma attack

with the appropriate pharmacological interventions without improvement in her clinical picture. By this time, it was noted that the patient had developed significant subcutaneous neck emphysema with suspected decreased breath sounds bilaterally. Tracheal deviation could not be adequately assessed because the trachea was still being controlled with the tracheal hook. The tracheostomy tube was immediately removed and an endotracheal tube was placed through the tracheostomy site with improvement in the oxygen saturation as well as the airway pressures. A chest film was then taken in the operating room by a radiology technician. However, before the film was returned, bilateral needle thoracoscopies were placed in the anterior chest wall. An instant rush of air was obtained and subsequently the patient could then be ventilated in a more satisfactory fashion with her oxygen saturation returning to the 90s. Another attempt to place the tracheostomy tube was made. In doing so, she again underwent a quick desaturation phase with an increase in airway pressures. A #7 armored endotracheal tube was then re-inserted again through the existing tracheostomy site. The endotracheal tube was positioned to the carina so as to avoid a significant leak around the cuff with pressures less than 30 mmHg. The stat chest film confirmed a tension pneumothorax. An immediate right chest tube was placed to ensure that the tension pneumothorax was adequately reduced. A left chest tube was also placed to treat her left pneumothorax. Multiple chest films indicated re-expansion of her lungs and confirmed that the position of the endotracheal tube was at the level of the carina. The patient was then transported back to the ICU in critical but stable condition.

After the operation, the patient remained on the ventilator in the ICU. Care was taken not to remove the chest tubes too soon because of the positive pressure mechanical ventilation. After an extended period of being placed on water seal, the chest tubes were eventually removed without incident. Because of the compromised position of the distal end of the endotracheal tube, multiple minor tube adjustments had to be frequently made to aid in maintaining ventilation to both lungs without creating too much of a air leak around the cuff. The patient had no obvious neurologic sequela from this event and she quickly returned to her pre-tracheostomy baseline.

She was taken back to the operating room for a bronchoscopy and tube change 5 days later. Bronchoscopy noted marked dilation and collapse of a large (>10 cm) segment of the anterior wall into the lumen of the trachea that was consistent with tracheomalacia. No tracheal perforation was noted. The length of the tracheomalacia was significant, extending down into the thoracic portion of the trachea just proximal to the carina. An attempt was made to place a #7 Portex, but it soon became apparent that in order to ventilate the patient, a ventilatory tube that would reach distal to her significant tracheomalacia would have to be placed. A #7 armored endotracheal tube was re-inserted. Tracheal reconstruction options were considered but not pursued. Multiple tube changes then took

place over the next few weeks. Progress was followed both clinically and with bronchoscopy. The patient remained

on the ventilator for 6 weeks before being successfully weaned. Bronchoscopy at the time of extubation showed

that the tracheal mucosa had sufficiently healed allowing the trachea to regain its structural integrity although a

decrease in diameter of the tracheal lumen was noted. The patient was discharged from the hospital one week later

on hospital day # 62.

Discussion

Tracheomalacia is classified as an extreme degree of dynamic compression of the airway consisting of a

decrease in cross-sectional area of the trachea by less than one-half of the expiratory compression. It is characterized

by abnormal tracheal collapse secondary to inadequate supporting cartilaginous and myoelastic elements. The

condition has been divided into congenital and acquired subgroups. Primary tracheomalacia is most likely due to

immaturity of the tracheobronchial cartilage[1] whereas acquired tracheomalacia is due to degeneration of previously

normal tracheal cartilages resulting from inflammatory processes, extrinsic vascular compression, bronchial

neoplasms, and tracheo-esophageal fistulas. [2]

By far the most frequent cause of acquired tracheomalacia is iatrogenic involving the endotracheal tube cuff

or the tracheostomy procedure.[2-4] Placement of an endotracheal tube in the adult airway is associated with well-

described complications resulting from injuries inflicted by the endotracheal tube. These injuries include traumatic

insertion, incorrect positioning of the tube, direct pressure from the tube itself, and direct pressure from the

endotracheal tube cuff. When overinflated, the endotracheal tube cuff places increased pressure on the mucosa of the

trachea, causing ischemic injury. The ischemia damages the tracheal mucosa and results in necrosis and infection at

the cuff site. This injury may eventually lead to the destruction of the tracheal cartilage.[5] Factors that may

contribute to the development of tracheal injury include the size of the tracheostomy tube, the presence of airway

infection, pre-existing lung disease, respiratory treatment and the use of steroids.[6]

Multiple factors played a role in the patient's tracheomalacia. Prolonged endotracheal intubation with high

cuff pressures (60-70 mmHg) was noted pre-operatively. Obtaining an adequate seal with normal cuff pressures was

not possible. Acceptable cuff pressures for endotracheal tubes are normally recommended to be less than 20mmHg

to decrease the chance of ischemic injury to the tracheal mucosa. [7,8] Judiciously increasing the cuff pressure was

done to achieve a seal, which led to iatrogenic injury to the tracheal mucosa and destruction of the tracheal cartilage.

Cuff pressures greater than 60mmHg pre-operatively should have been reason for suspicion that there was an

abnormality within the structural integrity of the trachea. In hindsight, after reviewing this patient's pre-operative chest x-rays, the existence of a large tracheal dilation was noted where the endotracheal cuff rests. Although chest X-rays have been shown not to be well correlated with ETT cuff pressures, the marked dilation may have suggested an abnormality (fig. 1A and 1B).[9] A bronchoscopy may have proven useful at this point.

The patient's history of COPD and previous intubations were also contributing factors to the development of tracheobronchial injury. Cinefluorographic studies of patients with COPD demonstrate an abnormally flaccid tracheobronchial tree that collapses upon expiration. Diminution in the diameter of these major branches of the airway produces central expiratory obstruction with consequent distal air trapping and alveolar overdistention.[10]

In addition, certain inhalation anesthetic, specifically Isoflurane, has been reported to induce tracheomalacia in patients without a history of respiratory problems.[3] A review of this patient's anesthetic record indicated that Isoflurane was used during the tracheostomy procedure. The use of Isoflurane anesthetic in this patient could have worsened an existing phenomenon. Katoh et al describe three cases of inhalation-induced tracheobronchomalacia.[3] All cases of inhaled anesthetic induced tracheomalacia resolved once the inhaled anesthetic was stopped.

The efficacy of placing an endotracheal tube in improving this patient's oxygen saturation appears to be a consequence of the tube's stenting effect on the semiflaccid trachea. The placement of a shorter tracheostomy tube, in comparison to the longer length of an endotracheal tube, led to acute desaturation with CO_2 retention and an increase in the airway pressure and eventually pneumothoraxes. There are reported cases of anesthetic complications as well as tracheostomy complications in which the collapsed trachea had to be mechanically opened either with insertion of an ET tube distally or with a bronchoscope. Katoh et al describes a case of tracheomalacia induced by Isoflurane anesthetic with abnormal respiratory patterns and desaturation that resolved with distal insertion of the ET tube.[3] Kang et al describes a tracheostomy complication that resulted in acute desaturation with high airway pressures and hypercapnia upon placement of a tracheostomy tube. They recommend temporary stenting of the airway by advancing the ET tube deeper to the carina.[11] Cogbill et al describes a tracheostomy complication in a newborn with primary tracheomalacia that resulted in cardiopulmonary arrest secondary to a collapsed trachea. They were able to reverse the arrest only with emergency bronchoscopy to mechanically open the collapsed trachea.[12]

The complication of pneumothorax associated with tracheostomy is well described in the literature. Three proposed mechanisms for this particular complication are examined by Berg et al.[13] One mechanism involves direct injury to the pleura. A second theory suggests that air dissection through the deep layer of the middle cervical fascia leads to pneumomediastinum ultimately resulting in a pneumothorax if the air ruptures into the mediastinal

pleura. A rupture of an alveolar bleb could explain the third mechanism that results in a pneumothorax. The incidence of pneumothorax ranges from 0-17%, depending on the age group studied.[14] Patients with a history of COPD and children have an increase risk of developing pneumothoraxes because the pleural domes are higher in the neck. Bilateral pneumothoraxes in this patient likely resulted from barotrauma from high airway pressures and air trapping.

Although a combination of etiologies is possible in this case…It is difficult to know whether the pneumothoraxes in this patient were indeed due to tracheomalacia or just secondary to surgical technique. Certainly, it is possible that there may have been a perforation outside of the lumen of the trachea by an ill-fitting tracheotomy tube while performing the tracheostomy, which could have caused a dissecting pneumothorax leading to this patient's rapid deterioration. The increase in airway pressures and pneumothoraxes could be explained this way. However, upon bronchoscopy, there were no perforations or tears in the tracheal wall. In addition, when the shorter tracheostomy tube was re-inserted, acute airway compromise returned with collapsed of the distal airway observed on bronchoscopy.

In severe tracheomalacia, there is loss of the structural support of the trachea with distension of the tracheal walls on inspiration and collapse of the trachea with expiration.[2] Consequently, there was an obstruction of the trachea resulting in CO_2 retention and desaturation. Placement of a longer endotracheal tube into the tracheostomy site acted as a stent for the collapsed airway allowing the patient to improve her ventilation. Theoretically, positive airway pressure should have been able to overcome the obstruction caused by the collapsed trachea. However, this was not observed in our case clinically and on bronchoscopy. Her clinical picture improved upon mechanically opening the distal trachea with a long endotracheal tube. Furthermore, it is important to note that the management of this patient included the use of frequent low-pressure cuff changes and the acceptance of a small amount of air leak to avoid progressive dilation of the trachea.

Tracheal resection with end-to-end anastomosis, tracheostomy, tracheoplasty with cartilage or dura graft, prosthetic stenting, and aortic or innominate artery suspension are some of the described treatment for severe tracheomalacia. Severity of disease varies greatly with etiology and type of obstruction. Mild cases can usually be managed conservatively. On the other hand, severe cases with insidious onset may require surgery. Fortunately, this patient's clinical outcome with conservative management was good, likely due to the acute nature of the injury.

In summary, a case of acquired tracheomalacia that appeared as a complication during a tracheostomy is presented. The clinical suspicion should be high when there has been a history of prolonged intubation with high

cuff pressure and air leak.   When this clinical situation is encountered, rather than increasing the cuff pressure, a diagnosis of tracheomalacia should be considered. A bronchoscopic exam should be performed to confirm the diagnosis. If the diagnosis is encountered during a tracheostomy, one of the immediate treatments may be to advance an endotracheal tube deeper, approximately to the position of the tracheal bifurcation. There have been a few cases of tracheostomy complications secondary to tracheomalacia reported in the literature. This case serves to remind us that the diagnosis of tracheomalacia relies on a variety of clues including history, clinical signs, radiographic exam, and clinical suspicion.

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Figure Legends

FIG 1A. Pre-admission chest film from one year previous showing normal tracheal anatomy.

FIG 1B. Chest film prior to tracheostomy showing significant dilation of the endotracheal tube cuff.