

Tracheal granulation as a cause of unrecognized airway narrowing

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Abstract

Tracheostomy is one of the most common elective surgical procedures performed in critically ill patients. The most frequent late complication after tracheostomy is the development of granulation tissue, a complication that may cause airway occlusion or result in airway stenosis. We report the successful management of a patient with tracheal granulation presenting as an unrecognised cause of difficulty breathing.

Key words: Airway, tracheal granuloma, tracheostomy

Introduction

Tracheostomy is one of the most common elective surgical procedures performed in critically ill patients.^[1] It can be associated with numerous complications, some of which continue to be relevant well after placement. The most frequent late complication is the development of granulation tissue, a complication that may cause airway occlusion or result in airway stenosis.^[2] We report the successful management of a patient with tracheal granulation presenting with difficulty in breathing.

Case Report

A 20-year-old man, presented to the emergency department (ED) with a history of difficulty in breathing through a tracheostomy tube (TT) since 2 days. Patient gave a history of a road traffic accident (RTA) 10 months ago, in which he sustained a head injury (diffuse axonal injury) which left

him quadriplegic and bed-bound. He required prolonged hospitalization and was tracheostomized 7 days after his RTA to facilitate prolonged mechanical ventilation. He was weaned off mechanical ventilation and discharged home with a metallic TT. Tracheostomy care and suction was done by caregivers at home. He was regularly followed up at a hospital nearby where his TT was changed every month. Four months prior to present admission to our hospital, the patient had difficulty breathing and was taken to the nearby hospital, where his TT was changed. He was mechanically ventilated for 2 days and discharged once stable.

The present episode also began with difficulty breathing. The patient's caregiver tried to change the TT but failed. The patient turned blue and was rushed to the nearby hospital. The doctor there attempted changing the TT but could not, but he successfully intubated the trachea with a 5-mm ID endotracheal tube (ETT) and referred him to our hospital for further management.

In the ED, his heart rate (HR) was 120/min, regular and blood pressure (BP) 110/70 mmHg. He was breathing at a rate of about 40/min despite being ventilated with an Ambu bag. Air entry was decreased bilaterally and expiratory phase was prolonged. A suction catheter could be passed down the ETT but encountered resistance distally. An arterial blood gas (ABG) showed pH 7.12, pCO₂ 69, pO₂ 320, HCO₃ 38, and saturation 99%.

He was shifted to the ICU but the oxygen saturation by the time he was admitted to ICU had fallen to 84–86%. An urgent bronchoscopy was done through the ETT which

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showed a collapsible, bleeding, soft-tissue mass causing near-complete obstruction of the trachea, with only a small hole visible during air movement into and out of the lungs. The mass was vascular, bleeding to touch, and the bronchoscope could not be guided beyond the obstruction. 2–3 ml of adrenaline 1:200,000 solution was sprayed through the bronchoscope which subsequently reduced the vascularity of the mass. Thereafter, the bronchoscope could be guided, though with some difficulty, through the visible hole. The growth was found to be about 1.5 cm long and beyond it the trachea was of normal calibre. The 5-mm ID ETT was pushed through the mass without significant bleeding. After this, the ventilation became considerably easier and the oxygen saturation picked up to about 94–96%. The patient was given dexamethasone 4 mg intravenous q6h for 24 h to reduce the edema. Computerized tomography (CT) scan confirmed luminal narrowing at the level of the sternoclavicular joint 4 cm from the carina with thickening of the posterior and right lateral walls of the trachea. Bilateral pneumothorax and extensive pneumomediastinum were also noted.

The granulation tissue was resected endoscopically the next day and the ETT was changed uneventfully to a distal-long stem TT to bypass the remaining obstruction. The patient was discharged next day in a satisfactory condition.

Discussion

A number of mechanisms can cause late complications after tracheostomy.^[3] Complications can be directly related to placement of the tube, leaving the tube in place for a prolonged period of time, or abnormal healing at the site of injured tracheal mucosa. They may be related to the inflated cuff of the TT or the tip of the TT, especially when it impinges on the posterior tracheal wall.^[2]

Granulation tissue may obstruct the airway at the level of the stoma and cause difficulty in replacing the TT if accidental decannulation occurs. Alternately, this granulation tissue can occlude tube fenestrations and lead to difficulty to decannulate. As the granulation tissue matures, it becomes fibrous and covered with a layer of epithelium. With the development of fibrosis, stenosis develops as the anterior and lateral aspects of the tracheal wall become narrowed at the level of the stoma.

Multiple risk factors are associated with stomal stenosis, including sepsis, stomal infection, hypotension, advanced age, male sex, steroids, tight fitting or oversized cannula, excessive tube motion (mechanical irritation), prolonged placement, and

disproportionate excision of anterior tracheal cartilage during the creation of the tracheostomy.^[4]

Suprastomal stenosis may occur when there is guidewire-related injury to the posterior tracheal wall with subsequent development of granulation tissue. Dilators used to enlarge the percutaneous dilatational tracheostomy stoma can injure the anterior tracheal cartilage (including tracheal ring fracture), causing these deformed structures to invaginate and protrude into the tracheal lumen, causing obstruction.^[5,6]

Tracheal stenosis may occur at the site of the tracheal-tube cuff, where ischemic injury to the tracheal mucosa can take place when cuff pressure exceeds the perfusion pressure of the capillaries of the tracheal wall. With prolonged ischemia, mucosal ulceration, chondritis, and cartilaginous necrosis may ensue, leading to the formation of granulation tissue. Shearing forces from the tube or the cuff may aggravate injury to the airway.^[2] Despite improvements in the design of tracheal tubes, however, tracheal stenosis after intubation remains an important cause of tracheal obstruction, which may be life threatening.^[7]

Tracheal stenosis may occur near the distal tip of the TT. Depending on the positioning of the tube, the tip may rub against either the anterior or the posterior tracheal wall. The latter may occur because standard TTs may be too short in patients with abundant soft tissue in the anterior neck. The resulting injury to posterior membranous wall may lead to stenosis or to trachea–esophageal fistula.

The granulation tissue in our patient may have been caused by prolonged mucosal irritation from the sharp edge of the metal TT and the trauma due to repetitive monthly changes of the tube.

Tracheal stenosis can occur while the patient remains mechanically ventilated. A retrospective study of 756 patients who had been ventilated for at least 15 weeks (3 weeks with an endotracheal tube followed by 12 weeks with a TT)^[8] showed that 37 patients (5%) developed failure to wean secondary to tracheal stenosis or obstruction from granulation tissue, often manifested as higher peak airway pressures or difficulty in passing a suction catheter.

An important aspect to diagnose tracheal stenosis early is to have a high index of suspicion, especially when a patient with a history of intubation or tracheostomy is under evaluation. Tracheal stenosis may produce no symptoms until the lumen has been reduced by 50–75%.^[4] Once the tracheal lumen has been reduced to ≤ 10 mm, exertional dyspnea occurs and if narrowed to ≤ 5 mm, dyspnea at rest or stridor may occur.

A CT scan provides precise information about the exact location and extent of the granulation/obstruction/stenosis. Laryngotracheoscopy or bronchoscopy should be done to define the exact site of stenosis and the length of the involved trachea and also rule out other coexisting pathology. History of inability to insert a TT or an adequate sized ETT, tachypnea, decreased air entry bilaterally, high airway pressures required to ventilate lungs, prolonged expiratory phase, and desaturation in the patient prompted us to do a bronchoscopy on him.

To prevent formation of granulation tissue, strategies have looked at avoiding excess mechanical irritation, placement of tubes of proper size and the use of swivel adapters and ventilator tubing support. Prevention of stomal infection is important, because infection may impair tissue healing. Meticulous stomal care is recommended to prevent bacterial contamination.^[9]

A number of therapeutic approaches have been described in patients with granulation tissue.^[2] In symptomatic patients, neodymium–yttrium–aluminum garnet (Nd-YAG) laser excision, with or without rigid bronchoscopic dilation may be the preferred approach. Suprastomal granulation can be excised by sharp dissection under bronchoscopic guidance. When a short web-like band of tracheal stenosis exists, laser excision can prove useful in reestablishing an adequate tracheal lumen with success rates as high as 60% after 1–3 sessions.^[10] In cases where laser resection is not feasible, stenting of airway or surgical repair are the best options.

A simple bronchoscopy procedure for tracheal stenosis had been developed by Freitag *et al.*^[11] for the classification of tracheobronchial stenosis. Treatment of granulation tissue depends on the underlying cause and the extent of the tissue. If a considerable amount of granulation tissue needs to be eliminated quickly, an option is to remove the tissue surgically with small instruments such as forceps or a soft tissue debrider, a tool that can cut the tissue and suction it away at the same time. Dilating the narrow part of the airway also is an option. If the need to remove the tissue is not as urgent, medication may be a treatment option. For example, in case of granulation tissue formation due to chronic infection, antibiotics may help minimize the tissue. At times, excessive secretions or saliva in the trachea can increase granulation tissue, so medications to dry up secretions can help to decrease the tissue. Medications to treat acid reflux, if present, may also reduce formation of granulation tissue in the airway.

Application of topical medications to shrink the granulation tissue can be effective too. These medications may include steroids, which can be applied directly to the tissue or

injected just beneath the granulation tissue. In addition, other medications decrease the number of fibroblast cells, which help to create granulation tissue. Tracheal stents have been used in the management of benign tracheal granulation tissue formation.^[12] An expandable metallic stent stops granulation or neoplastic tissue from proliferating through the mesh. Argon plasma coagulation and Tranilast have been successfully used to treat granulation tissue obstructing the airway after tracheal anastomosis.^[13] Endobronchial brachytherapy has been used successfully for long-term control of recurrent granulation tissue following tracheal stenosis.^[14] Mitomycin C (an antimetabolite known to inhibit fibroblast proliferation) has also been used for the treatment of tracheal cicatrix after tracheal reconstruction.^[15]

We report the successful management of a patient experiencing breathing difficulty with a TT *in situ*. The granulation tissue in our patient may have been caused by prolonged irritation by the sharp edge of the metal TT and the trauma due to repetitive monthly changes of the tube. Bronchoscopy played an important role in its early diagnosis and management.

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