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Journal of Otolaryngology-ENT Research

Case report

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Tracheal necrosis - a complication of percutaneous tracheostomy to remember

Abstract

Objectives: Tracheal necrosis is a rare but well-known complication of prolonged ventilation with a cuffed endotracheal tube. We report a case of iatrogenic tracheal necrosis from percutaneous tracheostomy (PT) leading to a critical airway and share our experience of managing this complication.

Methods: Case report with literature review.

Results: A 67year old Caucasian man underwent uneventful PT. He developed stridor after successful decannulation. The examination revelaed soft tissue prolapse into the tracheal lumen and 60% tracheal lumen narrowing. Exploration of the tracheostomy site revealed necrosis of anterolateral wall of the trachea. The defect was regarded too large to be amenable to simple reconstruction. A tracheostomy tube was inserted to secure the airway and allow scar contracture to take place. The tube was downsized gradually allowing eventual successful decannulation.

Conclusion: Injury to the tracheal cartilage following PT can lead to localized infection, ischaemia and necrosis resulting in a compromised airway.

Keywords: Intubation; Percutaneous tracheostomy; Tracheoesophageal fistula; Tracheal necrosis; Cartilage; Surgical tracheostomies; Extubation; Bronchial alveolar lavage; Adrenaline; Nasolaryngoscopy; Bronchoscopy

Volume 3 Issue 4 - 2015

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Received: October 13, 2015 | **Published:** October 29, 2015

Abbreviations: BAL, bronchial alveolar lavage; MRSA, methicillin resistant staphylococcus areus; VISA, vancomycin-intermediate staphylococcus aureus; TCF, tracheocutaneous fistula

Introduction

Tracheostomy is a commonly performed intervention with several benefits in the treatment of patients with chronic respiratory failure. Refinement of the tracheotomy technique, using percutaneous approaches have allowed bedside tracheostomy placement in the modern intensive care unit. Both percutaneous and surgical tracheostomies are associated with several early and late complications.[1,2] These complications are generally well documented in the literature. Our case reports an unusual scenario of iatrogenic tracheal necrosis from percutaneous tracheostomy (PCT) leading to a critical airway. In this article, we share our experience in the management of this condition and review of recent literature.

Case report

A 67year old man was admitted to casualty after a house fire. Although he had sustained minor burns to his body, there was soot around his nose and mouth along with singed eyebrows. The patient was prophylactically intubated. After resuscitation he was transferred to ITU where he remained overnight. The following morning he failed extubation and required re-intubation. The decision to proceed with percutaneous tracheostomy was made in an attempt to wean him off the ventilator. Flexible bronchoscopy was performed and a bronchial alveolar lavage (BAL) was sent for microbiology analysis. The BAL culture had grown Methicillin Resistant Staphylococcus Areus (MRSA). MRSA was also isolated in the patient's blood and tracheostomy swab cultures. The patient was treated with Teicoplanin following microbiologist advice.

Over several weeks of ventilatory support, the patient was slowly weaned off the ventilator. This was partly due to patient's comorbidities of chronic obstructive pulmonary disease. After 51days he was decannulated and transferred to the Respiratory ward. A week later, he developed acute stridor in the ward. He was treated with steroids and nebulised adrenaline on the diagnosis of tracheal stenosis secondary to granulation tissue. An ENT and anaesthetic review was urgently sought. Flexible nasolaryngoscopy on the ward revealed a flail segment of tissue from the tracheostomy site protruding into the tracheal lumen. A second swab of the tracheal secretion was taken and MRSA was isolated. Further microbiology advice was sought and this was treated with prolonged course of antibiotic. The patient subsequently had an exploration surgery of his upper airway and trachea. A 5x5mm tracheo-cutaneous fistula was discovered. Bronchoscopy showed loss of the anterior and right anterolateral walls of the trachea worst affecting the rings 1 and 2. A tracheostomy was temporarily placed with an aim to reconstruct the trachea at 6 to 9months. However, his condition was successful managed by debridement and serial downsizing of the tracheostomy tube with eventual decannulation.

Discussion

The incidence of major tracheal complications secondary to endotracheal intubation ranges from 0.3 to 19%. This includes tracheitis, tracheomalacia,[3] tracheoesophageal fistulas, tracheo-innominate artery fistula,[4] cuff trachiectasis, tracheal stenosis[5] and tracheal necrosis.[1,2] Similarly, these complications can be seen with tracheostomy.[6] However, tracheal necrosis leading to tracheal perforation is a rare but serious complication of percutaneous tracheostomy and its management is often dependent on the severity of the tracheal damage, presence of any co-existing structural involvement and patient's comorbidities.

Tracheal damage can start as early as 3 -5days after intubation. The cascade of injury stems from initial local tracheal ulceration followed by necrosis, which may progress to tracheal stenosis and in more severe cases; it may progress to full-thickness perforation through the tracheal wall. This mechanism of tracheal damage at cuff site has been demonstrated in both autopsy studies and investigation in animal models.[7,8] Overinflation of the endotracheal cuff beyond 20-40cm H2O could also cause further insult and ischemia of the tracheal vasculature[9] leading to tracheomalacia, tracheal dehiscence and perforation. Routine measurement of endotracheal cuff pressures has not yet been implemented in many ICUs. While direct manometry remains the only reliable method of ensuring therapeutic cuff pressures, some centers suggest chest films in the intubated patient may prove a useful adjunct for identifying at-risk patients before they manifest potentially catastrophic complications. Alternatively, a high-volume low pressure cuff has also been used to reduce the incidence of cuff-related complications.[10]

Histologically, foreign body reaction begins as wound healing process with accumulation of exudate at the site of injury, infiltration of inflammatory cells, and subsequent formation of granulation tissue. However, with the presence of a foreign body, this inhibits the process of full healing by preventing the reabsorption and reconstruction that would normally occur in wound healing leading to chronic inflammation and fibrosis.[11] In a systematic review and meta-analysis consisting of 1212 patients, clinically important wound infection was diagnosed in 6.6% of patients with PCT based on data from 11 randomised controlled trials.[12] Persistent infection of the stoma is rarely seen with PCT and was only reported in 1 patient in a cohort study consisting of 800 mixed ICU patients who underwent PCT.[13] A history of persistent stoma site infection, and in this case, the presence of MRSA despite treatment with teicoplanin, vancomycin-intermediate S. aureus (VISA) and vancomycin-resistant S. aureus (VRSA) should be suspected. Vancomycin and teicoplanin is glycopeptide antibiotic which has been used for over 3 decades to treat serious methicillin-resistant Staphylococcus aureus infections. The increased frequency of multidrug-resistant bacteria, especially vancomycin-resistant strains, has focused interest on three new semi-synthetic lipoglycopeptides for the treatment of infections caused by gram-positive bacteria: oritavancin, dalbavancin, and telavancin. These agents contain a heptapeptide core, common to all glycopeptides, which enables them to inhibit transglycosylation and transpeptidation (cell wall synthesis). In addition to inhibiting cell wall synthesis, telavancin and oritavancin are also able to disrupt bacterial membrane integrity and increase membrane permeability; oritavancin also inhibits RNA synthesis. All the lipoglycopeptides demonstrate potent in vitro activity against Staphylococcus aureus and Staphylococcus epidermidis regardless of their susceptibility to meticillin, as well as Streptococcus spp. Both dalbavancin and telavancin are active against vancomycin-intermediate S. aureus (VISA), but display poor activity versus vancomycin-resistant S. aureus (VRSA). Oritavancin is active against both VISA and VRSA.[14] However, the phase III trials evaluating oritavancin for the treatment of acute bacterial SSSIs have yet to publish their results.[15]

Tracheal stenosis commonly occurs either in the area of the stoma after de-cannulation or where the cuff has been inflated.[1] The incidence of post-tracheostomy tracheal stenosis has been described as between 8 percent[6] and 31 percent[16] with varying degree of severity. It is a very common finding post-tracheostomy as a degree of stenosis occurs with natural granulation tissue. However, only a small number (3-12%) present with clinical features.[16,17] Tracheostomy associated fistula, on the other hand, is uncommon and only present in <1% of cases.[11] This is often associated with stoma site infection and chronic inflammation in the surrounding tissue as observed in our patient. Tracheal necrosis is considerably rare but cases have been reported in post-intubation[7] and following radiotherapy.[18]

The management of tracheal perforation has been extensively described ranging from placement of tracheal stents to more extensive tracheal resection and reconstruction.[6,19,20] The initial management aim to minimise tracheobronchial soilage by placing a tracheostomy tube with a cuff below the site of the fistula. In the presence of communication with the oesophagus, gastric reflux content need to be managed aggressively by replacing any existing nasogastric tube with a draining grastrostomy and adequate nutritional support is facilitated by inserting a feeding jejunostomy tube. However, majority of the cases will require surgical closure as spontaneous closure of tracheoesophageal fistula is rare. This is usually delayed until patient's condition is stable and weaned from mechanical assisted ventilation as positive pressure ventilation after tracheal repair carries an increased risk of anastomotic dehiscence and restenosis.[20,21] The most effective treatments are oesophageal bypass and oesophageal stenting. Bypass is demonstrated to resolve respiratory soilage and allow fairly normal swallowing, but it should be reserved for patients who can tolerate a major operation. Stenting can be offered to nearly all patients regardless of their physiologic condition. Stenting also limits aspiration and allows swallowing. Esophageal exclusion is rarely indicated in the current era of familiarity with stenting techniques. Direct fistula closure and fistula resection do not often yield satisfactory results.[21] Tracheal resection and reconstruction has been described for more severe and problematic tracheal stenosis and in recent years, the use of a tubed pectoralis major myocutaneous flap for reconstruction after extended circumferential resection of trachea due to necrosis.[22] As for the surgical management of persistent tracheocutaneous fistula (TCF), several approaches have been advocated in the literature. Drezner and Cantrell performed a 5year review of their technique and outcome of TCF closure and proposed that patients with large TCF (a defect of the anterior tracheal wall of > or = 4mm diameter), can be successfully treated with excision of the fistula, replacement of the tracheotomy tube and healing by secondary intention after a short recannulation period.[23] In more recent years, Pallua and Wolter had described using a tunneled supraclavicular artery island flap incorporating fascia or bone to reconstruct the anterior tracheal wall. In their series of 12 patients, this method appears to provide good functional and aesthetic outcome.[24] Other methods have been described using dermal inter-positional fat graft,[25] transplantation of an ear cartilage graft and deltopectoral flap.[26] Nevertheless, the success of any treatment is dependent on correction of any airway contamination and malnutrition. In our case, the patient had a 5mm x 5mm TCF and was managed successfully with serial debridement and downsizing of the tracheostomy tube.

Conclusion

In the modern hospital setting necrosis of the tracheal cartilage secondary to infection of a tracheostomy site must be considered among other differential diagnosis of tracheal stenosis after decannulation. Injury to the tracheal cartilage is best avoided by careful entry into the tracheal lumen through the soft tissue between the tracheal rings and avoiding fracturing the tracheal ring. Such injury can lead to localized infection, ischaemia and necrosis resulting in a compromised airway. In select cases, a large necrotic tracheal wall segment can be successfully managed by debridement and serial downsizing of the tracheostomy tube with eventual decannulation.

Citation: Shakeel M, Keh S, Walker A, et al. Tracheal necrosis -a complication of percutaneous tracheostomy to remember. *J Otolaryngol ENT Res.* 2015;3(4):319–321. DOI: 10.15406/joentr.2015.03.00076

Summary

Percutaneous tracheostomy is commonly performed in patients admitted to intensive care units requiring long term ventilation. Tracheal necrosis is a very rare complication of PT. Fracture of the tracheal ring during PT can lead to localized infection, ischaemia and necrosis of the tracheal cartilage. Patients presenting with stridor after decannulation should be suspected to have soft tissue prolapse in to the tracheal lumen through a necrosed tracheal wall. In select cases, a large necrotic tracheal wall segment can be successfully managed by debridement and serial downsizing of the tracheostomy tube with eventual decannulation.

Acknowledgments

None.

Conflicts of interest

Author declares there are no conflicts of interest.

Funding

None.

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