Atresia of the Trachea Following Repeated Percutaneous Dilational Tracheotomy*

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Percutaneous dilational tracheotomy (PDT) and conventional tracheostomy are still competing methods to provide an airway for intensive care patients requiring assisted ventilation. Tracheal stenosis is a late complication for any tracheostomy and long-term intubation. However, late complications in PDT have not been extensively studied. This article is the first to report on total atresia of the subglottic larynx and cervical trachea after PDT. The dimension of the lesion is visualized by three-dimensional reconstructed CT scan. The etiology of this condition is discussed.

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Key words: percutaneous; tracheal stenosis; tracheostomy; tracheotomy

Abbreviation: PDT = percutaneous dilational tracheotomy

Percutaneous dilational tracheotomy (PDT) is a relatively new technique1 and has replaced conventional tracheostomy for long-term intubated patients in many ICUs. The reported advantages of PDT are that it is simpler, more quickly accomplished, and more cost-effective than conventional tracheostomy.2–4 Several different techniques exist for percutaneous tracheostomy, but Rapidtrac (Surgitech Medical Pty. Ltd.; Sydney, Australia) and PDT have been the most extensively studied. Perioperative complication rates of PDT are comparable to those of conventional tracheostomy.5 However, only few randomized, controlled trials have evaluated the safety of PDT.3,4 Perioperative complications of PDT include desaturation and false insertion, but fatal intratracheal bleeding6 and tension pneumothorax7 have also been reported. Postoperative complications involve hemorrhage, tube displacement, subcutaneous emphysema, stoma infection, vocal cord paralysis, and tracheoesophageal fistula.8 Tracheal stenosis is an important late complication after tracheostomy and usually develops 2 to 12 weeks after decannulation.8 The incidence of symptomatic tracheal stenosis after conventional tracheostomy occurs in 1 to 8% of the cases reported.9,10 Late complications from PDT have not yet been studied extensively. A few studies, all of which consist of a small series of cases, have reported a zero to 5% occurrence of tracheal stenosis following PDT.9,11,12

Complete atresia of the subglottic region and the cervical trachea as a serious complication of PDT has not yet been reported in the literature.

CASE REPORT

A 59-year-old woman was admitted to a general hospital with the acute onset of increasing dyspnea. Her medical history was significant for COPD. Respiratory insufficiency required orotracheal intubation of the patient. A chest radiograph revealed a right tension pneumothorax requiring immediate needle decompression and tube thoracostomy. After 4 days, the pneumothorax significantly improved and the tube thoracostomy could be removed. However, pneumonia with the Proteus organism required prolonged intubation and intensive antibiotic therapy. Therefore, after 10 days of intubation, a PDT was performed by an experienced surgeon. The technique described by Ciaglia et al9 was performed at the third tracheal interspace under bronchoscopic control. Assisted ventilation was used throughout the intervention. Improvement of the respiratory situation after 2 weeks allowed the tracheal tube to be removed. Unfortunately, 4 days later an unexpected relapse into respiratory insufficiency and pneumonia occurred, requiring a renewed PDT. Bronchoscopic control revealed no tracheal stenosis at that time. Finally, after 2 more weeks, the respiratory situation allowed definitive removal of the tracheal tube. However, short-term closing of the tracheostoma caused severe dyspnea. Therefore, the patient was suspected of having a tracheal stenosis and was referred to the department of otolaryngology.

To determine the exact localization and length of the stenosis, the patient underwent microlaryngoscopic evaluation that revealed a normal glottic region with some granulation tissue in the posterior subglottic space (Fig 1, top) and a complete subglottic atresia somewhat caudally (Fig 1, bottom). During open tracheotomy, further examination confirmed the lack of any tracheal lumen above the tracheostoma. CT scans (Fig 2) of the neck and three-dimensional reconstruction (Fig 3) demonstrate these findings.

Further operative evaluation revealed that resection of the stenosis and end-to-end anastomosis was not feasible since the stenosis reached high in the subglottic larynx. Other reconstruction techniques (eg, laryngotracheal reconstruction with cartilage grafting) were considered inadequate for the patient, since it was believed that she would not tolerate aspiration, which is sometimes encountered following such procedures. Therefore, it was decided that the patient would have to remain tracheotomy dependent, which remains the condition to date.
Discussion

Percutaneous tracheostomy has been used for > 10 years as an alternative to conventional open tracheostomy. Different techniques exist, but PDT is the most popular and well-studied method. The main indication is the need for ventilation in intensive care patients. The procedure gives the ICU doctors the possibility to perform tracheostomy by themselves without organizing efforts such as transporting the patient to an operating theater and coordinating with other specialists. The incidence of perioperative and postoperative complications, specifically under bronchoscopic control, are not significantly higher than conventional tracheostomy, although several fatal complications have been reported.6,7,12–14 The few studies
investigating the frequency of late complications of the PDT method reported a low incidence of tracheal stenosis.\textsuperscript{11,15} An important factor related to the complication frequency of PDT is the experience of the surgeon with this method; several authors have reported a significant learning curve for PDT.\textsuperscript{15} Furthermore, technical details of the PDT method should be noted.\textsuperscript{16} van Heurn et al\textsuperscript{17} emphasized that surgeons should not perform PDT at the subcricoid level, as well as avoiding oblique insertion of the cannula, which can lead to a protrusion of the anterior tracheal wall. In our case report, PTD was performed at the third tracheal interspace by a surgeon experienced with this technique. The mechanisms that caused this critical outcome remain unclear. However, it can be hypothesized that three different mechanisms may have contributed to the final trachea atresia. First, a postoperative infection probably caused by the Proteus organism may have occurred, leading to destruction and necrosis of the cartilaginous rings. Second, a fracture of one or more tracheal rings during PDT may have happened, causing major mucosal tears and consecutive cicatricial obliteration of the tracheal airway. Third, the trachea may have been torn off completely at the tracheostoma level, probably because the respiratory situation of the patient required urgent airway management on the occasion of the second PDT due to impending respiratory distress. Since the bronchoscopic examination during the second PDT revealed no tracheal stenosis or luminal pathology, it is likely that in this case the second PDT intervention was the significant contributor to tracheal damage. Repeated PDT intervention may be more risky and difficult due to altered anatomy, concurrent infection, and frequent trauma. No studies are yet available dealing with the complications of repeated PDT. However, in cases requiring continual extubation and intubation, a stable and epithelial tracheostoma may have advantages. This case demonstrates that this minimally invasive approach can lead to the total destruction of the trachea.

REFERENCES


Figure 3. Three-dimensional reconstruction of the CT scan showing the lack of any airway from the subglottic to the tracheostoma level.
A case report is presented of spontaneous chylothorax successfully treated by conservative means. The helpful role of the inhibitory peptide, octreotide, is discussed. (CHEST 2001; 119:964–966)

Key words: chylothorax; octreotide

Abbreviation: TPN = total parenteral nutrition

Since thoracic duct ligation was introduced in 1948 by Lampson1 for the surgical treatment of chylothorax, no new invasive or noninvasive definitive therapy has been available.2 We report the first case of prompt cessation of lymphorrhea in an adult patient with chylothorax using octreotide, a long-acting somatostatin analog.

CASE REPORT

A 79-year-old woman was admitted to St. Mary Hospital in Hoboken, NJ, complaining of progressively debilitating weakness and dyspnea. Her non-Hodgkin’s lymphoma had been in remission with chemotherapy off and on for > 8 years. She admitted having a heavy sensation in her chest. She was alert, and the only physical findings were the absence of breath sounds and dullness to percussion on the left chest. On chest radiography, she had almost complete opacification of the left hemithorax. Laboratory tests revealed hemoglobin level of 8.5 g/dL. The serum albumin level was 2.9 g/dL on admission and 1.7 g/dL on the 25th day after admission. The chest fluid culture revealed no growth. Chemical analysis of the chest fluid revealed cholesterol, 89 mg/dL, and triglycerides, 640 mg/dL. Cytologic smear of the fluid showed granulocytes, reactive mesothelial cells, and copious lymphocytes highly suggestive of lymphoproliferative disease. ECG indicated a “borderline ECG.” CT scan revealed large nodes at the left thoracic inlet and confluent periaortic adenopathy from diaphragm to pelvis.

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Progressive deterioration of the patient’s strength and nutrition occurred from the massive loss of proteins in spite of total parenteral nutrition (TPN) of 1,800 to 2,200 calories daily. The serum albumen level kept decreasing (Fig 1). Eleven days after admission, doxycycline, 300 mg, was instilled into the left chest. The fluid became watery and loculated, and the original chest tube clotted, necessitating a second tube thoracostomy to drain the watery, cloudy fluid (Fig 1).

Octreotide, 100 μg tid, was started subcutaneously, and the chest drainage stopped by the third day. Treatment was continued for a total of 17 days (Fig 1). Oral intake was withheld from the seventh to the 28th day of hospitalization. The patient required a thoracentesis on the 29th day to empty the loculated, yellow, watery fluid. The patient became stronger and much less dyspeptic. The albumen level started improving. The left chest remained clear until discharge on the 45th day of hospitalization. She has remained well for > 20 months at home.

COMMENT

It has been a decade since the arrest of lymphorrhagia in the neck was observed after the use of somatostatin.3 Nine years later, chylothorax was treated by the somatostatin analog, octreotide, in a 4-month-old boy.4 It took 2 days for the lymphorrhea to stop in the first patient and 11 days in the second patient. In our patient, the lymphorrhea stopped by the third day.

When thoracic duct ligation was first proposed in 1948, the mortality associated with chylothorax was reduced from 50 to 15%.1 Most patients now are given a 14-day course of TPN. Approximately 20 to 50% of patients will then require surgical treatment with a mortality of 15%. Tumor, usually of lymphatic type, is responsible for the chylothorax in adults.

Patients with pulmonary lymphangiomatosis, a rare condition in young women, fatal usually from chylothorax, may benefit from octreotide therapy. Octreotide reduces the thoracic duct flow and its triglyceride level. Side effects, such as diarrhea or dizziness, thrombocytopenia, hepatotoxicity, and other reactions, did not occur. The drug has been successfully used in the treatment of GI fistulae.5

Octreotide has pronounced inhibitory effect on basal and pentagastrin-stimulated gastric acid secretion. It blocks pancreatic secretion by inhibiting enzyme secretion. It also inhibits biliary secretion. It decreases the volume of high-output GI fistulae, thus lessening the metabolic systemic derangements of the patient. In fistulae of lesser output, the cessation of drainage is dramatic and curative.

Since the GI secretory volume and enzymes are decreased by the octreotide, it is thus logical to expect a decrease in the volume and protein content of the fluid in the thoracic duct. The therapeutic implication, therefore, follows that the volume and protein loss in the hydrothorax may be reduced to the point that the leakage heals.

It is true that cessation of oral intake stops food and fluid absorption from the GI tract. It also stops the stimulation of GI secretions into the gut. Less volume is thus offered for absorption and flow into the thoracic duct.